

For your information-

A LIST OF
COW & GATE
WORLD-FAMOUS PRODUCTS

COW & GATE MILK FOOD—"Cow's milk made safe and suitable for babies"

BRESTOL—"The Humanised Cream"

COW & GATE HALF CREAM—"For babies unable to digest full cream"

COW & GATE SEPARATED MILK—"For babies unable to digest fat"

COW & GATE CHOCOLATE MILK—"Nature's tonic beverage"

PEPTALAC—"The instant predigested food"

HEMOLAC—"The Iron milk food"

LACIDAC—"The lactic acid milk"—in three strengths

FRAILAC—"For frail and premature babies"

LACQUIN—"The tone food of the Tropics"

ALLERGILAC—"For Allergy in Infancy"

PROLAC—"The Protein Milk"

SPRULAC—"For the dietetic treatment of Sprue"

NATURAL TEAT—"Designed to secure an inflow of milk by the same muscular action as in breast feeding"

ENAMEL MEASURE—"Graduated in ozs., pints and tablespoons"

COW & GATE RUSKS. COW & GATE FEEDERS
COW & GATE "GRIPON" TEATS

WRITE
FOR
LITERATURE



AND
CLINICAL
SAMPLES

© 467

COW & GATE LTD., GUILDFORD, ENG.



If you specify Gee & Watson Ltd. for your process blocks of medical work, you will be certain that all that extremely fine detail which the photograph shows will be fully retained.

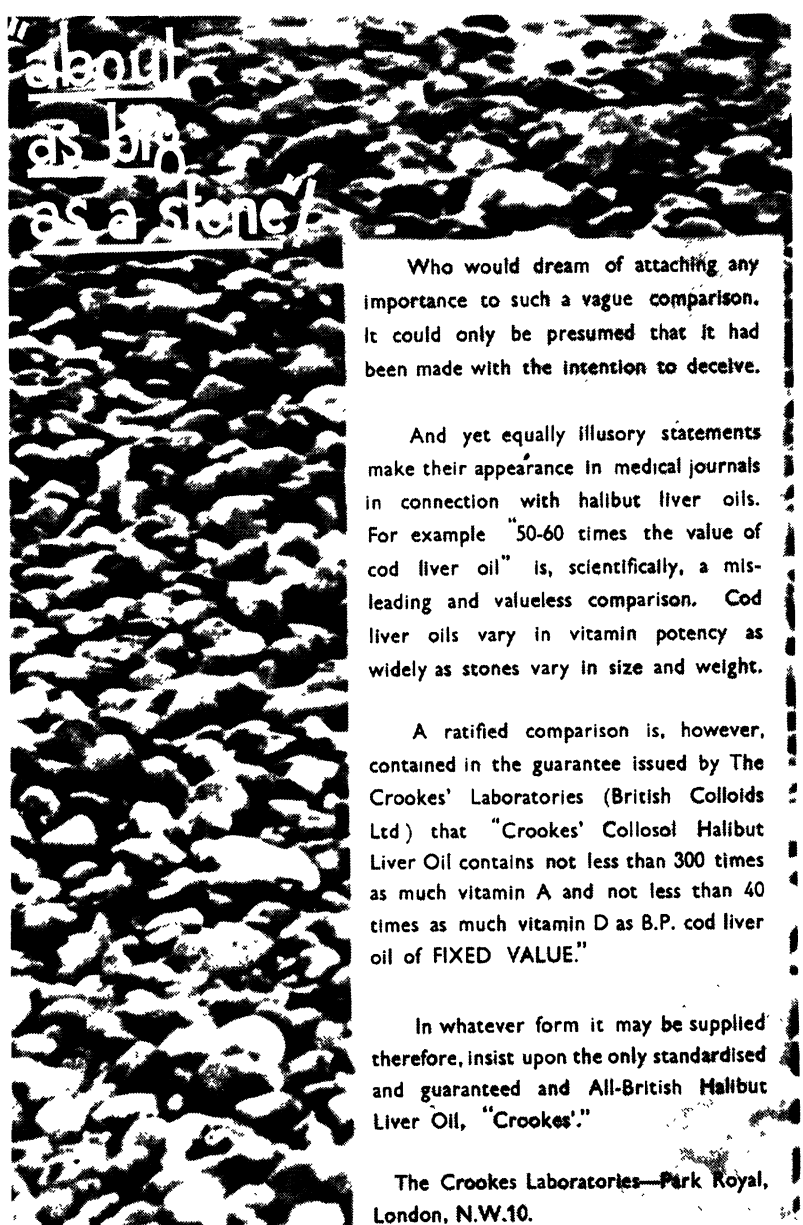
We have specialised in the production of these blocks for 30 years, and have an unbroken connection with some of the medical publishers right throughout that period.



GEE & WATSON LTD

Fine Art Process Engravers • Telephone: Central 7337-8-9

111 SHOE LANE, FLEET STREET, LONDON, E.C. 4



about
as big
as a stone/

Who would dream of attaching any importance to such a vague comparison. It could only be presumed that it had been made with the intention to deceive.

And yet equally illusory statements make their appearance in medical journals in connection with halibut liver oils. For example "50-60 times the value of cod liver oil" is, scientifically, a misleading and valueless comparison. Cod liver oils vary in vitamin potency as widely as stones vary in size and weight.

A ratified comparison is, however, contained in the guarantee issued by The Crookes' Laboratories (British Colloids Ltd) that "Crookes' Collosol Halibut Liver Oil contains not less than 300 times as much vitamin A and not less than 40 times as much vitamin D as B.P. cod liver oil of FIXED VALUE."

In whatever form it may be supplied therefore, insist upon the only standardised and guaranteed and All-British Halibut Liver Oil, "Crookes'."

The Crookes Laboratories—Park Royal,
London, N.W.10.

HEWLETT'S "COMPLETE G.P." BAG



Size 13½ in. 6½ in. x 9 in.

Introduced and manufactured only by

C. J. HEWLETT, & SON, Ltd., 35 42, Charlotte St., LONDON, E.C.2

THIS bag is so arranged that every item is immediately visible and accessible the moment the lid is lifted. The front of the bag need only be let down to get at the compartment holding the emergency dressing, etc. The large central space is to hold a folding stethoscope, and room is left for further instruments such as sphygmomanometer, etc.

PRICE, with Instruments and Fittings, in Black or Brown Cowhide, lined with Rexine **£7 0 0**

PRICE, in Black Rexine, instead of Leather **£6 10 0**

A fully illustrated pamphlet, with suggested list of drugs for inclusion in the bag, will be sent on application

Where an AID is indicated for YOUR DEAF PATIENT . .

MIDHUNT'S
Ardent
FOR DEAF EARS

is Your **SAFE** Prescription

BECAUSE it is backed and fully guaranteed by the world's greatest hearing service. Being individually fitted to the needs of each particular case from a wide range of types, "ARDENT" can be relied upon for every type of deafness where an aid is indicated, from "hard-of-hearing" to acutely deaf. In addition, an after-fitting service throughout the country meets the needs of changing conditions.

Medical prescriptions made up to the minutest detail

SPECIAL for MEDICAL MEN

"ARDENT" Stethoscope is specially designed for those members of the medical profession who are deaf; those now in use are reported to be giving excellent results. Several unique features in this and other special apparatus for medical men

No expense is incurred until the Patient is tested and satisfied



THE WORLD'S GREATEST HEARING SERVICE

Send for full details and Medical Reports. Free Home Tests arranged for Doctors and Patients.

309 OXFORD ST., LONDON, W.1.

(Coloured Building with Square Clock.)

Mayfair 1380/1718.

CARDIFF, MANCHESTER, BIRMINGHAM, HULL, NEWCASTLE, BRISTOL, DUNDEE, LIVERPOOL, LITCHFIELD, EXETER, LEEDS, LONDON, GLASGOW

Process Blocks by

NOAKES

can be relied upon for consistent quality and faithful reproduction of detail in all Medical Subjects.

For a considerable number of years we have been responsible for the Process Work in the "Medical Press & Circular" and in addition, many of the illustrations appearing in "Modern Treatment in General Practice" have been prepared by us.



MEDICAL SUBJECTS FAITHFULLY REPRODUCED

NOAKES BROTHERS LIMITED,
Process Engravers,
16 New St. Sq., London, E.C.4.
Telephone : Central 6851 (15 lines).

BAYER PRODUCTS IN GENERAL PRACTICE

- CIRCULATORY - 'ICORAL' New Analeptic
FAILURE 'PADUTIN' Peripheral dilator
'LACARNOL' Vaso-regulator
- OBESITY - 'ELITYRAN'
Standardised Thyroid Full Gland Extract
- MIGRAINE - 'LUMINAL'
'CAFINAL' 'IMPLETOL'
- EPILEPSY - 'LUMINAL'
'PROMINAL' (with reduced hypnotic effect)
- RHEUMATISM - 'NOVALGIN'
LUMBAGO Frequently effective when salicylates fail
Better tolerated
- ANÆMIAS - 'CAMPOLON'
Liver extract (intramuscular)
Potent, safe, easy to administer
- COMMON COLD 'GARDAN'
"Hot Lemon and 'Gardan' "
- PNEUMONIA - 'OMNADIN'
Non-specific immunising therapy
" 'Omnadin' in any case "
-



Write for clinical supply and literature to:

BAYER PRODUCTS LTD.
AFRICA HOUSE, KINGSWAY
LONDON, W.C.2

MODERN TREATMENT IN GENERAL PRACTICE

Edited by

CECIL P. G. WAKELEY, D.Sc.,
F.R.C.S., F.R.S.E.

EDITOR OF "THE MEDICAL PRESS AND CIRCULAR"

BUTTERWORTH & CO. (INDIA) LTD.

CALCUTTA - - 6, Hastings Street (P.O. Box 251)

BOMBAY - Jehangir Wadia Bldg., Esplanade Road

MADRAS - - 317, Linga Chetty Street

PRINTED IN GREAT BRITAIN

PREFACE

THIS book is born of popular demand. A series of articles entitled "Modern Treatment in General Practice" was started in THE MEDICAL PRESS AND CIRCULAR. These proved immediately acceptable; in fact back numbers went out of print one after another before all those who desired them could be supplied. It was, therefore, thought that a re-issue of these articles in book form, together with an equally popular series, the Quarterly Symposiums, would be welcomed. In a few cases articles of special interest outside these series have been included.

The articles of which this volume is composed are written for the general practitioner by the specialist, and are designed and intended to set forth concisely and interestingly the latest progress made in medicine and surgery, with special stress on diagnosis and treatment. They cover a wide range of subjects, and an equally extensive range of thought, for the contributors have been drawn from almost every school and every part of the country.

A fellow editor reviewing one of the Symposium numbers of THE MEDICAL PRESS AND CIRCULAR wrote: "It must be a very rare occurrence for a practitioner not to find in each number something of immediate practical importance to him. The editors, when any new discovery is established, always get an article by some sound authority on the subject . . . The Quarterly Symposiums are a very useful addition to the practitioner's library." This is a fair summary of the editorial policy of the Journal and a certain guide to the practical value of the contents of this volume, which we hope will become recognised as a useful addition to the general practitioner's library.

CECIL P. G. WAKELEY,
Editor.

8, HENRIETTA STREET,
LONDON, W.C. 2

LIST OF ARTICLES AND CONTRIBUTORS

		PAGE
1. TREATMENT OF DIABETIC COMA	George Graham, M.D., F.R.C.P., Physician, St. Bartholomew's Hospital.	1
2. ACUTE CIRCULATORY FAILURE	D. Evan Bedford, M.D., F.R.C.P., Physician to Out-Patients at the Middlesex Hospital and the National Hospital for Diseases of the Heart.	8
3. HEAD INJURIES IN CHILDREN	Cecil P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E., Senior Surgeon, King's College Hospital, and West End Hospital for Nervous Diseases.	15
4. MODERN TREATMENT OF BURNS AND SCALDS AND OTHER USES OF THE 2 PER CENT. TANNIC ACID COMPRESS	Philip H. Mitchiner, M.D., M.S., F.R.C.S., Honorary Surgeon to H.M. The King; Surgeon with charge of the Septic Wards, and Senior Surgeon to Out-patients, St. Thomas's Hospital, London.	20
5. OBESITY IN WOMEN	Ethel Browning, M.D., Assistant Pathologist, Pickett-Thomson Research Laboratory, St. Paul's Hospital, London.	26
6. OBESITY IN MEN	Ivo Gelkie Cobb, M.D., M.R.C.S., Physician, Norwich Union Insurance Company.	32
7. THE MODERN TREATMENT OF EARLY SYPHILIS	M. Sydney Thomson, M.A., M.D., F.R.C.P., F.R.S.E., Physician for Diseases of Skin to King's College Hospital and the Belgrave Hospital for Children.	38
8. JAUNDICE IN INFANCY AND CHILDHOOD	Reginald Lightwood, M.D., M.R.C.P., D.P.H., Assistant Physician for Diseases of Children to the Westminster Hospital.	46
9. THE ÆTIOLOGY AND TREATMENT OF DISSEMINATED SCLEROSIS	R. S. Allison, M.D., M.R.C.P., Assistant Physician, Royal Victoria Hospital, Belfast; Consulting Physician, Benn Ulster Eye, Ear and Throat Hospital.	51
10. BILIARY COLIC	Charles Newman, M.A., M.D., F.R.C.P., Assistant Physician, King's College Hospital; Assistant Registrar, Royal College of Physicians.	58
11. BLOOD TRANSFUSION	Norman Hill, M.D., M.R.C.P., Physician to the Metropolitan Hospital; Senior Physician to the Belgrave Hospital for Children.	65
12. SUDDEN INSANITY	Edward Mapother, M.D., F.R.C.P., F.R.C.S., Medical Superintendent, Maudsley Hospital; Physician for Psychological Medicine, King's College Hospital.	72
13. MIGRAINE AND ITS TREATMENT	Fergus R. Ferguson, M.D., D.P.H., F.R.C.P., Assistant Physician, Royal Infirmary, Manchester; Lecturer in Clinical Medicine, Victoria University, Manchester.	83
14. VERTIGO AND ITS TREATMENT	W. Russell Brain, D.M., F.R.C.P., Assistant Physician to the London Hospital and the Royal London Ophthalmic Hospital	94

✓ 15. MINOR SURGERY OF THE EYE	N. Bishop Harman, M.A., M.B., F.R.C.S., Consulting Ophthalmic Surgeon, West London Hospital.	103
✓ 16. THE TREATMENT OF PELVIC FLOOR PROLAPSE	Thos. G. Stevens, M.D., F.R.C.S., F.C.O.G., Obstetric Surgeon, St. Mary's Hospital, London; Surgeon, The Hospital for Women, Soho Square, London.	112
✓ 17. COCCAL INFECTIONS OF THE URINARY TRACT	Clifford Morson, O.B.E., F.R.C.S., Surgeon to St. Peter's Hospital for Urinary Diseases.	118
✓ 18. THE TREATMENT OF OBSTRUCTIVE JAUNDICE	Charles Newman, M.D., F.R.C.P., Junior Physician, King's College Hospital, London; Assistant Registrar, Royal College of Physicians.	121
✓ 19. THE TREATMENT OF PLEURISY	J. F. Halls Dally, M.A., M.D., M.R.C.P., Senior Physician to the St. Marylebone General Dispensary.	125
✓ 20. PLEURAL EFFUSIONS — SEROUS AND PURULENT	A. J. Scott-Pinchin, M.D., F.R.C.P., Physician, City of London Hospital for Diseases of Heart and Lungs.	131
✓ 21. SEASONAL HAY - FEVER AND ITS TREATMENT IN THE BRITISH ISLES	George W. Bray, M.B., Ch.M., Asthma Research Fellow, Hospital for Sick Children, Great Ormond Street, London.	137
✓ 22. THE EPILEPSIES IN CHILDHOOD	W. G. Wyllie, M.D., F.R.C.P., Physician to Out-patients, Hospital for Sick Children, Great Ormond Street, London.	146
✓ 23. FLAT FOOT	E. P. Brockman, M.Ch., F.R.C.S., Orthopædic Surgeon, Westminster Hospital.	153
✓ 24. MODERN TREATMENT OF PYORRHOEA	A. T. Pitts, D.S.O., M.R.C.S., L.R.C.P., L.D.S., Dental Surgeon to the Royal Dental Hospital and the Middlesex Hospital; Examiner in Dental Surgery to the Royal College of Surgeons.	159
✓ 25. THE TREATMENT OF THE COMMON COLD	Terence East, M.D., F.R.C.P., Physician, King's College Hospital and Woolwich Memorial Hospital.	168
✓ 26. THE TREATMENT OF SEA-SICKNESS	Surgeon - Commander A. Vavasour Elder, D.S.C., V.D., M.R.C.S., L.R.C.P., R.N.V.R. (retired); Surgeon to the White Star Line.	172
✓ 27. INSOMNIA AND ITS TREATMENT	E. B. Strauss, M.A., D.M., M.R.C.P., Assistant Physician to the Cassel Hospital for Functional Nervous Disorders.	178
✓ 28. OTITIS MEDIA IN CHILDREN	E. Watson - Williams, M.C., Ch.M., F.R.C.S., Surgeon in Charge, Ear, Nose and Throat Department, Bristol Royal Infirmary.	189
✓ 29. THE TREATMENT OF RHEUMATOID ARTHRITIS IN GENERAL PRACTICE	W. S. C. Copeman, M.A., M.B., M.R.C.P., Physician to British Red Cross Society's Clinic for Rheumatism, Peto Place; Assistant Physician, Children's Department, West London Hospital, and Hospital of St. John and Elizabeth; Consultant, L.C.C.	199

30. INFECTIONS OF THE HAND	R. M. Handfield - Jones, M.S., F.R.C.S., Surgeon to St. Mary's Hospital, London.	
31. THE TREATMENT OF DI- GESTIVE DISTURBANCES IN THE BREAST-FED INFANT	Robert E. Stean, M.D., F.R.C.P.I., Physician to the Meath Hospital, and to the National Children's Hospital, Dublin.	211
32. THE TREATMENT OF DIAR- RHOEA IN INFANTS	Rowland Hill, M.D., M.R.C.P., Physician to the Hospital for Sick Children, Belfast.	219
33. THE MODERN TREATMENT OF NASAL CATARRH	A. L. Yates, M.C., M.D., F.R.C.S., Assis- tant Surgeon, Ear and Throat Depart- ment, Prince of Wales' Hospital.	224
34. CONGENITAL HYPERTRO- PHIC STENOSIS OF THE PYLORUS	Cecil P. G. Wakeley D.Sc., F.R.C.S., F.R.S.E., Senior Surgeon to King's College Hospital; Hunterian Professor, Royal College of Surgeons of England.	232
35. SUBACUTE COMBINED DE- GENERATION OF THE SPINAL CORD	T. Kay MacLachlan, M.A., M.B., F.R.F.P.S., Assistant Physician, Glas- gow Royal Infirmary.	239
36. CÆLIAC DISEASE	Stanley Graham, M.D., Visiting Physi- cian, Royal Hospital for Sick Children, Glasgow.	248
37. PINK DISEASE	A. Victor Neale, M.D., M.R.C.P., Assistant Physician, Children's Hos- pital, Birmingham; Assistant Physi- cian, Queen's Hospital, Birmingham.	255
38. THE CLINICAL SIGNIFI- CANCE OF ERYTHEMA NODOSUM	W. R. F. Collis, M.A., M.D., F.R.C.P., D.P.H., Physician to the National Children's Hospital; Pædiatrician to the Rotunda Hospital, Dublin.	265
39. TREATMENT OF ANÆMIA IN INFANCY	Hugh W. Josephs; from the Harriet Lane Home of the Johns Hopkins Hospital, and the Department of Pædiatrics, Johns Hopkins University, Baltimore, Maryland, U.S.A.	271
40. THE TREATMENT OF SOME OF THE COMMONER SKIN DISEASES CAUSED BY COCCI	R. M. B. MacKenna, M.D., M.R.C.P., Honorary Dermatologist, Liverpool Stanley Hospital.	278
41. THE TREATMENT OF CON- JUNCTIVITIS	W. C. MacFetridge, M.D., Ophthal- mic Surgeon, Sir Patrick Dun's Hospital and Drumcondra Hospital, Dublin.	286
42. THE TREATMENT OF PNEU- MONIA IN CHILDREN	T. R. R. Todd, M.D., F.R.C.P., Physician, Leith Hospital; Assistant Physician, Royal Infirmary, Edinburgh.	291
43. THE TREATMENT OF TUBERCULOUS DISEASE OF THE HIP	H. F. MacAnley, M.Ch., F.R.C.S.I., Assistant Surgeon, Mater Misericordiæ Hospital, Dublin.	297
44. THE SPA TREATMENT OF CHRONIC RHEUMATISM	W. S. C. Copeman, M.A., M.B., M.R.C.P., Hon. Physician, B.R.C.S. Clinic for Rheumatism; Assistant Physician, West London Hospital (Children's Dept.) and Hospital of St. John and Elizabeth; Consultaut, L.C.C.	303

45. SOME COMMON EYE DISEASES IN ADULTS	A. H. Levy , F.R.C.S., Surgeon to Central London Ophthalmic Hospital.	309
/ 46. SOME COMMON EYE DISEASES IN CHILDREN	A. H. Levy , F.R.C.S., Surgeon to Central London Ophthalmic Hospital.	316
/ 47. THE COMMON SKIN DISEASES OF CHILDHOOD	M. Sydney Thomson , M.A., M.D., F.R.C.P., F.R.S.E., Physician for Diseases of Skin, King's College Hospital and Belgrave Hospital for Children.	323
48. MODERN METHODS OF EXPOSING THE BRAIN AND ITS MENINGES	Lambert Rogers , M.Sc., F.R.C.S., F.A.C.S., Assistant Director of the Surgical Unit, the Welsh National School of Medicine, The Royal Infirmary, Cardiff.	331
✓ 49. THE TREATMENT OF ACUTE ABDOMINAL CONDITIONS IN CHILDREN	R. A. Ramsay , M.Ch., F.R.C.S., Senior Surgeon to the Metropolitan Hospital, and to the Belgrave Hospital for Children.	337
✓ 50. FRACTURES OF THE ANKLE - JOINT AND THEIR FACILITIES	Geraldine Barry , M.S., F.R.C.S., Assistant Surgeon, Royal Free Hospital.	349
51. A PRACTICAL SURVEY OF SPA FACILITIES	G. V. Worthington , M.B., B.Ch., Llandrindod Wells.	356
✓ 52. THE ÆTIOLOGY AND TREATMENT OF ANÆMIAS DUE TO DEFICIENCIES	L.S.P. Davidson , B.A., M.D., F.R.C.P.E., Regius Professor of Medicine, University of Aberdeen.	366
/ 53. CHRONIC HOARSENESS	Herbert Tilley , B.S., F.R.C.S., Consulting Surgeon (Diseases of Ear, Nose and Throat), University College, Hospital, London.	379
/ 54. CLINICAL FEATURES AND TREATMENT OF ELECTRICAL INJURIES	Hilary Long , B.Sc., M.B., B.S., Diploma of University of Vienna; Travelling Student of the University of London, 1932-33.	390
/ 55. COMMON FUNGUS INFECTIONS OF THE SKIN	C. Howard Whittle , M.A., M.D., M.R.C.P., Physician to the Skin Department, and Clinical Pathologist, Addenbrooke's Hospital, Cambridge.	398
/ 56. PIGMENT FORMATION IN THE SKIN	G. H. Percival , M.D., Ph.D., F.R.C.P., D.P.H., Lecturer on Diseases of the Skin, Edinburgh University; Assistant Physician to the Department for Diseases of the Skin, Royal Infirmary, Edinburgh.	405

CHAPTER I.

TREATMENT OF DIABETIC COMA

BY

GEORGE GRAHAM, M.D., F.R.C.P.

Physician, St. Bartholomew's Hospital.

THE great majority of patients suffering from diabetes mellitus used to die in coma, but since the use of insulin the number has been very much decreased. In many cases the task of saving the patient is an easy one, but in some it is very difficult, and demands the unremitting attention of doctor and nurses.

The best treatment for coma is the prevention of its onset, and it is important to consider the conditions under which it may develop. Coma may be precipitated by the following causes :

(1) The development of an acute disease. This may be (*a*) easy to diagnose, *e.g.*, a carbuncle, a cellulitis, or appendicitis ; (*b*) difficult to diagnose, *e.g.*, a deep-seated abscess, pyrexia without any physical signs, vomiting. In some cases the signs of the disease develop after the patient has recovered from coma. (2) The omission of the usual dose of insulin for twenty-four hours. (Coma only occurs when the daily dose is forty or more units.) (3) The taking of large amounts of carbohydrate, sufficient to cause marked hyperglycæmia for many months.

The onset of coma can often be prevented if the patient is seen soon after the first symptoms appear. This is especially the case when the coma is due to the occurrence of an acute illness. Thus, a patient who usually passes no sugar at all may wake in the morning with fever and lack of appetite, and may develop coma within twenty-four hours if insufficient insulin is given. The insulin is often omitted altogether because the patient associates food and insulin, and, if he has no appetite for his breakfast, is afraid to take insulin. Insulin should always be given, and the dose can be gauged by the following rules : If the urine contains no sugar, one-third of the usual dose should be given ; if the urine

contains a little sugar, half the dose ; if much sugar, the full dose. If the patient is well enough to take the usual amount of carbohydrate, the dose of insulin should be increased by five units. The size of the next injection must be decided either after an estimation of the blood sugar six hours later, if this is possible, or by the examination of the urine, which should be collected and tested for sugar every three hours. The urine in the first three hours after the insulin will contain sugar, and the urine four to six hours after the insulin will also contain sugar if the dose of insulin was not adequate. Another dose of insulin should then be given, and its size can be determined by the following rules : If the Benedict test is green, half the first dose should be given ; if it is yellow, the same dose ; if it is red, double the dose. The same procedure is followed every six hours until the urine in the four to six hour period is free from sugar. When this occurs no insulin is given until sugar re-appears, and then only half the last dose is given. Special forms for charting the results of the urine tests are in use at St. Bartholomew's Hospital, and are of great assistance in watching the course of the disease.

The onset of coma should be prevented by this method, but the task is made much easier if the blood sugar is estimated at frequent intervals.

A diabetic patient may become comatose because he has had either too little or too much insulin. Fortunately, the two conditions can be easily distinguished. In insulin coma : (1) the skin is usually very white, but may be normal in colour ; (2) the breath does not smell of acetone ; (3) the respirations are shallow ; (4) the eyeball tension is normal or raised ; (5) the urine is usually sugar free, but may contain it if the bladder has not been emptied for some hours, as the sugar may have been excreted when the blood sugar was above 200 mg. per 100 c.c. ; (6) the urine will not contain aceto-acetic acid, but may do so if the bladder has not been emptied for some hours (if the patient has taken salicylates, the ferric chloride test will give a dark-purple colour ; Rothera's nitro-prusside test is not affected by any drug, and is much more reliable) ; (7) the blood sugar is below 70 mg. per 100 c.c., and may be as low as 40 mg. per 100 c.c.

In diabetic coma : (1) The skin is usually flushed ; (2) the breath smells of acetone ; (3) the respirations are deep (this is shown not only by the movements of the chest, but also of the

abdomen, and the latter are most characteristic of the condition); (4) the eyeball tension is much lower than usual; (5) the urine always contains large amounts of sugar; (6) the urine always contains large amounts of aceto-acetic acid; (7) the blood sugar is over 200 mg. per 100 c.c., and may be very high—500 to 800 mg. per 100 c.c.

Initial Treatment.—This should be started as soon as the diagnosis is made and the blood collected for the estimation of the sugar content. A large dose of insulin should be given, and the actual size depends on (1) the severity of the condition, (2) the usual dose of insulin.

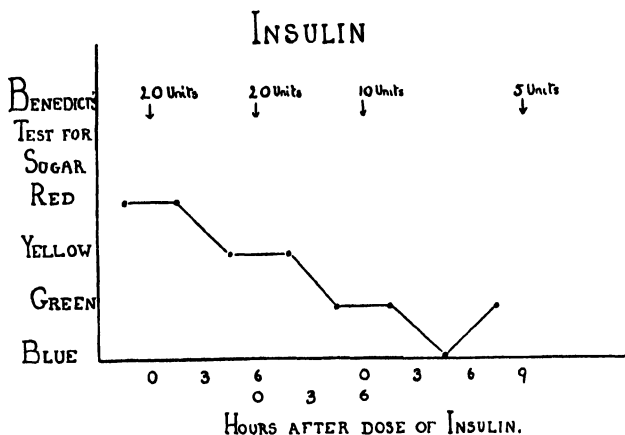


FIG. 1.—Shows the sugar in the urine and the dose of insulin which should be given during an infection.

(1) If the patient is not deeply comatose the initial dose should be 50 units, but if the coma is very deep, 70 or over 100 units should be injected. (2) If it is known that the patient usually takes large doses of insulin, say, 40+40 units, the initial dose should be at least half as great as the doses recommended for the ordinary cases, *e.g.*, 75 instead of 50, and 110 and 150 instead of 70 and 100 units.

The blood sugar is nearly always very high in cases of coma; but sometimes it is only moderately raised, *e.g.*, 300 mg. per 100 c.c. It is therefore recommended that 50 g. (2 oz.) of sugar should be given at the same time as the insulin to reduce the risk of hypoglycæmia. The sugar should be given by mouth in 10 oz. of water

if the patient can swallow, but if he cannot swallow it should be given by a nasal catheter or œsophageal tube, and in this case it should be dissolved in 20 oz. of water. It is very important to give adequate fluid, and if the patient can swallow, 10 oz. of water should be given every hour for the first nine to twelve hours, however quickly the patient recovers. If the patient is very deeply comatose, the fluid should be given intravenously, 600 c.c. every three hours until recovery occurs.

It is not necessary to give sodium bicarbonate to the great majority of patients in diabetic coma, but when the patient is deeply comatose, 600 c.c. of a 2 per cent. solution of sodium bicarbonate should be given, together with the 50 g. of dextrose. There is no need to repeat the dose of sodium bicarbonate, as the 12 g. should be sufficient, and if further doses of dextrose are required the sugar should be dissolved in distilled water.

Patients are sometimes very cold and suffer from "shock." They should be warmed as quickly as possible, and the best method is by means of a hot-air bath, or, if this is not possible, by means of hot bottles, but the latter method is very inefficient. A hot-air bath can be improvised in the following way: Two cradles are placed over the bed, and two or more blankets put over them. A chair with a high back is placed about two feet from the end of the bed, and the blankets brought over the chair. A small electric radiator or oil stove is placed in the space between the bed and chair, and the hot air passes over the patient; the risk of fire is negligible.

Patients are often very constipated, and a dose of castor oil, one to two ounces, should be given with the first dose of sugar.

A careful search should be made for any gross source of sepsis. Superficial abscesses should be opened soon, but an operation for an appendix abscess, for example, should be delayed until the general condition has improved.

Signs of Improvement.—These are: (1) Gradual recovery of consciousness; (2) decrease in depth of respiration, so that the abdominal breathing becomes less marked; (3) increase in the eyeball tension; (4) improvement in the volume of the pulse and a rise in the blood pressure.

Subsequent Treatment.—The effect of the first dose of insulin must be carefully watched, if possible by estimations of the blood sugar about three or six hours later, and always by the urine tests.

The urine should be collected every three hours, and if necessary a catheter should be passed. It is better not to tie in the catheter unless a St. Mark's apparatus is used, because of the grave risk of causing cystitis, and so adding to the patient's troubles during convalescence.

A very deeply comatose patient is unlikely to show much improvement in three hours, and it is necessary in these cases to repeat the same dose of insulin and sugar. This should be given intravenously because of the failure to improve.

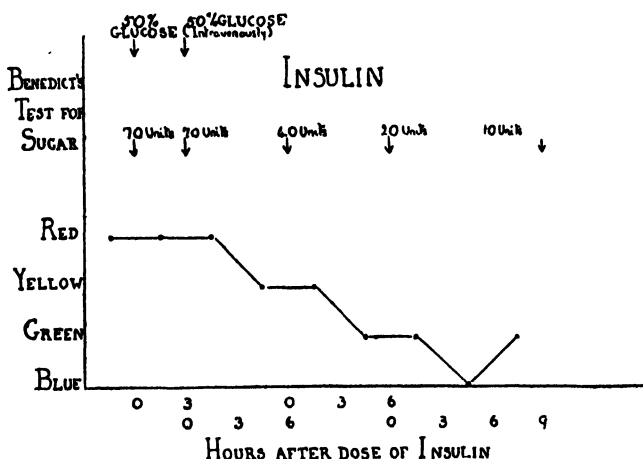


FIG. 2.—Shows the dose of insulin and sugar which should be given in a very severe case of diabetic coma which shows no improvement at the end of the third hour. There is no need to give any insulin at this stage if the patient shows definite improvement.

The patient usually shows some sign of recovery in three hours after a dose of insulin, but the urine will, of course, contain much sugar, and it is usually unnecessary to give any insulin at this time. At the end of six hours the second specimen of urine is examined. Another dose of insulin should be given if this specimen contains sugar, and the size should be determined according to the following rules: If the patient is better, half the initial dose should be given; if he is about the same, the initial dose should be repeated; if he is worse, the dose should be half as much again. A dose of 50 g. of sugar should always be given in the last instance, and it is probably safer to give it in any case at the end of the sixth hour.

The same procedure should be adopted after the second dose of insulin, and the patient will probably show definite signs of recovery by this time. It is, therefore, usually unnecessary to consider giving any insulin until the result of the four to six hour period is known. If no sugar is present, and the patient is definitely better, no insulin should be given until the sugar reappears in the urine, and then half the preceding dose should be given. If sugar is present in the four to six hour period, and the patient is definitely better, half the preceding dose should be given. If, however, the patient is still comatose, insulin should be given according to the same rules as at the end of the first four to six hour period. Thus, a patient who was given 80 units on admission and 120 units six hours later, should now be given 180 units if there are no signs of improvement. The same procedure is continued every six hours until the patient recovers consciousness. When this occurs, the possibility of hypoglycæmia must be considered.

Signs of Overdose.

The danger of giving too much insulin, and so causing a severe attack of hypoglycæmia, is a real one, and the symptoms should be known by all the nurses and attendants. The usual signs of hypoglycæmia are lacking, as the patient is not well enough to appreciate hunger and lassitude. Sweating is an uncertain sign, as it may occur for other reasons. The most usual sign is a relapse into deep coma after a partial recovery of consciousness. The condition can be diagnosed by the great change in the respiratory movements, which are now shallow instead of deep, the abdominal respiration being practically absent; the eyeball tension is now normal instead of very low; the face is usually pale; the breath should not smell of acetone, but may still do so if a change from hyper- to hypo-glycæmia has been a rapid one. The urine may also contain sugar and acetone bodies for the same reason. Some patients have fits, and the limbs may become spastic. The symptoms all call for prompt treatment, and the diagnosis should be made without waiting for the result of a blood sugar. One c.c. (ten units) of pituitrin, or, if this is not available, 1 c.c. of adrenalin, should be injected at once, and this should be followed by 50 g. of dextrose. The sugar should be given by a nasal catheter or œsophageal tube, as the patient will be unable to swallow. Preparations should be made for an intravenous injection of 300 c.c. of

20 per cent. dextrose, and this should be given if the patient has not improved in fifteen minutes. This treatment will raise the blood sugar, and the patient will rapidly recover consciousness. If it is a severe hypoglycæmia, insulin should not be needed for at least six hours, and the dosage must be small and controlled by the blood sugar and testing of the three-hourly specimens of urine.

The cases which do not recover seem to die of circulatory failure, and drugs do not help the heart at all. The giving of plenty of fluid seems to prevent the circulatory failure from occurring. Patients in coma usually recover with sufficient insulin and sugar, provided that the patient is seen early enough. The case must be regarded as an emergency, and the patient's condition must be considered every three or six hours.

CHAPTER II.

ACUTE CIRCULATORY FAILURE

BY

D. EVAN BEDFORD, M.D., F.R.C.P.

Physician to Out-Patients at the Middlesex Hospital and the National
Hospital for Diseases of the Heart.

ACUTE circulatory failure may be defined as a state of collapse due to a sudden diminution in the output of the heart, associated with a fall in blood pressure. In addition, there are certain forms of acute engorgement of the lungs which must be classed as varieties of circulatory failure. The normal cardiac output depends on an adequate venous return and on an efficient ventricular contraction. A diminished output may result either from a failure of the venous return or from an inefficient ventricular systole. Thus there are two distinct forms of circulatory failure (the peripheral vascular type, in which the venous return fails, and a central cardiac type, in which the propulsive force of the ventricles is impaired.) In one case the heart receives no blood to pump, in the other it is unable to pump the blood it receives. A combination of these two factors often occurs. Any sudden cardiac accident that weakens the ventricles—for example, coronary thrombosis—may provoke a reflex vaso-motor paralysis, while a fall in blood pressure of peripheral origin soon reacts adversely on the heart by diminishing the coronary flow.

Peripheral Vascular Failure.

The underlying cause of this type of failure is paralysis and dilatation of the peripheral vessels, which allows the circulating blood to drain into the splanchnic area. The blood pressure falls, and the heart is deprived of blood. This vascular paralysis may originate in the nervous centres, as in syncope, or may be due to a direct toxic action of some histamine-like substance on the vessels themselves. Peripheral failure is seen typically in surgical shock, after severe injuries or hæmorrhage, and in diabetic coma.

Its main features are low blood-pressure, a small, rapid, regular pulse, intense pallor, cold extremities, sweating, small contracted veins, cerebral anæmia, and profound prostration. There is no need to dwell on the familiar picture of surgical shock, but certain forms of spontaneous vascular failure must be mentioned, because they are not infrequently mistaken for true heart attacks.

Fainting.—The ordinary fainting-fit is a form of transient vascular failure, the main feature of which is cerebral anæmia and loss of consciousness. Sir Thomas Lewis has shown that the mechanism underlying a faint consists of a sudden vaso-dilatation with fall of blood pressure, together with an increase of vagal tone, resulting in a slowing of the pulse to 40-60 a minute. Loss of consciousness is not usually sudden, as in epilepsy, but is preceded by a premonitory phase during which the blood pressure is falling, and symptoms such as giddiness, pallor, coldness and limpness of the limbs, visual disturbances, nausea and sweating occur. The pulse becomes smaller and slows to 60 or below, and eventually consciousness is lost, when the pulse may disappear at the wrist. The unconscious stage lasts usually for a few minutes, but occasionally it is more prolonged, and there is slight muscular twitching. Recovery is gradual, and a low blood-pressure with some degree of faintness may last for hours.

Fainting is commonest in young subjects with healthy hearts, though it also occurs in older people and in those with organic heart disease, especially aortic regurgitation and hypertension. The attacks are often provoked by prolonged standing, fatigue, crowded and overheated places, and by emotional upsets, and are more likely to occur during ill-health and at the menstrual periods. Fainting almost always occurs in the standing posture, and never in bed or during sleep, an important point of distinction from epilepsy.

Vaso-vagal Attacks (Gowers' Syndrome).—Though Lewis has applied the term "vaso-vagal" to ordinary faints, it was originally used by Gowers to describe a particular form of syncopal attack which is clinically quite distinctive. Vaso-vagal attacks have been clearly described by Collier and others, and must be familiar to all who encounter many cases of fits and faints in the course of practice. I believe that the mechanism of the attack is the same as in the case of fainting, for a fall of blood pressure and a slowing of the pulse always occur. But the process is more gradual in

onset and far more prolonged, and there are premonitory psychical disturbances comparable with an epileptic aura. The attack is heralded by a feeling of lassitude and a peculiar sense of remoteness from the outside world. The surroundings seem vague and unreal, and restlessness and anxiety may develop. Soon increasing pallor is evident, the limbs feel cold, there is epigastric discomfort, and a sensation of respiratory difficulty. The pulse slows and becomes smaller or scarcely palpable, a state of collapse develops, and finally consciousness may be lost. A condition of intense prostration may last for an hour or more, during which consciousness is impaired and actual syncope may recur. In some cases there is a great anxiety and distress.

Like faints, these attacks tend to occur periodically in those predisposed to them. They are most often seen in middle-aged subjects of unstable nervous type. They occasionally occur at night in bed like epilepsy, and I have seen severe diarrhoea and abdominal distention usher in an attack. The premonitory psychical disturbances, the gradual onset and prolonged fall of blood pressure are the distinctive features of vaso-vagal attacks. The collapsed condition of the patient during prolonged attacks is often alarming, yet recovery is invariable. The diagnosis depends on a history of previous attacks, on complete recovery from each attack, and on the absence of signs of organic heart disease.

Ménière's syndrome is sometimes accompanied by a fall of blood pressure, pallor, and even collapse, but the characteristic sense of rotation, and the association with deafness and tinnitus, is diagnostic. The possibility of a cerebral lesion and of epilepsy must always be kept in mind as a cause of sudden loss of consciousness.

Treatment of Peripheral Failure.—The aim of treatment is to increase the volume of circulating blood, to restore vascular tone, and to diminish cerebral anæmia. Warmth is the first essential. The foot of the bed should be raised, and bandaging of the limbs is of value. If there has been hæmorrhage, blood transfusion is indicated, otherwise intravenous saline or glucose can be given.

Hypertonic glucose, 50–100 c.c. of a 30 per cent. solution, is probably most efficient. (As the heart is not primarily at fault, drugs of the digitalis group are useless. Of drugs acting on the vessels, adrenalin and ephedrine are most certain. Strychnine is less effective, and a dose of at least $1/12$ grain is required. Fainting requires no special treatment beyond lowering of the head, the

application of warmth or friction to the limbs, and a dose of brandy. In prolonged faints and vaso-vagal attacks, $\frac{1}{2}$ c.c. of 1 in 1,000 adrenalin, or half a grain of ephedrine, should be injected, and repeated if necessary. Cardiazol-ephedrine, 1 c.c., is also effective. Between the attacks, sedatives rather than stimulants, are beneficial, and bromides or luminal are most suitable.

Acute Cardiac Failure.

In the past, sudden circulatory failure arising in the heart was commonly attributed to "acute dilatation." The electro-cardiogram has provided the true explanation of many forms of acute cardiac failure, showing that (they arise from the inception of an abnormal rhythm.) Radiological observations lend no support to the conception of acute dilatation of the heart as a primary cause of failure. Rapid changes in the size of the heart are exceptional, and dilatation is observed as the result of continued failure rather than as a cause of acute failure.

Sudden impairment of the functional capacity of the ventricles is often due to an abnormal rhythm, which drives the heart either too fast or too slowly for efficient pumping. Ventricular fibrillation, a state of inco-ordinate muscular twitching amounting to paralysis, is probably the cause of sudden death in many cases of angina and coronary thrombosis, during chloroform anæsthesia and in digitalis poisoning. Sudden ventricular failure may manifest itself as syncope, dyspnoea, pain, or palpitation, and the clinical varieties may conveniently be grouped according to the predominant symptom.

Cardiac Syncope.—Any abrupt fall of blood pressure, if maintained, will lead to cerebral anæmia, and may induce loss of consciousness. Occasionally syncope occurs at the onset of coronary thrombosis. More frequently it is due to a disturbance of rhythm. Any form of paroxysmal tachycardia, if the rate is excessive, may lead to syncope, though if the heart is sound, the attacks are usually well tolerated. Rarely, in cases of auricular flutter, the ventricles respond at times to each auricular beat, attaining a speed of 250–300 a minute, when syncope may occur. A history of attacks of rapid beating, starting and ceasing abruptly, should lead to a correct diagnosis. Syncope from too slow a rate of beating is known as an Adams-Stokes attack. In some cases of heart-block there are periodic pauses in the ventricular beating, during which the pulse

ceases, the blood pressure falls, and consciousness is lost. Apart from long pauses in the pulse, syncopal attacks are liable to occur whenever the ventricular rate falls below twenty a minute. Short periods of ventricular standstill cause faintness, giddiness, and transient pallor, but in longer pauses there is loss of consciousness, often accompanied by muscular twitching. The association of syncopal attacks with a very slow heart rate should always suggest Adams-Stokes syndrome, and an electrocardiogram should be obtained. In cases of partial block, the pulse rate may be normal between the attacks. The treatment of an Adams-Stokes attack consists of injecting subcutaneously $\frac{1}{2}$ c.c. of 1 in 1,000 adrenalin, which increases the ventricular rate, and often stops the attacks. The injection may be repeated if the pulse rate falls again.

In syncope from ventricular fibrillation, either at the onset of coronary thrombosis or during anæsthesia, the heart can sometimes be restarted by an intracardiac injection of adrenalin. A long needle is pushed through the fourth intercostal space, keeping close to the sternum, until the cavity of the right ventricle is reached and blood withdrawn, when 1 c.c. of adrenalin is injected. In cessation of the heart during anæsthesia, if adrenalin fails, massage of the heart through an abdominal incision should be tried, while artificial respiration is maintained.

Cardiac Failure with Pain.—Coronary thrombosis is the most important cause of collapse with pain. In a typical case there is a severe and prolonged anginal seizure, accompanied by shock, ashen pallor, sweating, fall in blood pressure, subnormal temperature, and often vomiting, followed perhaps by dyspnœa, œdema of the lungs and congestive failure. The pulse may be impalpable, is usually small, rapid and regular, but occasionally normal in rate, or irregular. Slight fever and rarely pericardial friction succeed the pain. Instead of pain there may be syncope, or severe dyspnœa combined with collapse. The diagnosis is not usually difficult, but in a doubtful case an electrocardiogram often proves conclusive.

The first step in treatment should be the relief of pain by morphia, injecting at least a quarter of a grain at once, and repeating it if the pain is not dulled. Absolute rest and warmth are essential, and sips of brandy may be comforting. Nitrites are useless and dangerous, and strychnine is of little value. Digitalis is only required in special cases where congestive failure or auricular

fibrillation develop. Glucose should be given by mouth, and intravenous injection has been used. Oxygen is indicated if there is dyspnoea or cyanosis. Once the acute painful stage is over, complete rest in bed for at least six to eight weeks should be insisted on.

Acute Cardiac Dyspnoea.—Cardiac “asthma” is the commonest form of sudden cardiac dyspnoea. It is a sign of left ventricular exhaustion, and occurs especially in (hypertensive) heart disease, less often in coronary disease or syphilitic aortitis. In spite of the name, the attacks consist simply of rapid breathing without bronchial spasm, (except in asthmatic subjects.) There is reason to believe that sudden pulmonary engorgement provokes the attacks, and some degree of pulmonary oedema nearly always develops. In hypertensive cases the blood pressure remains elevated, or even rises at the onset of dyspnoea, an important diagnostic point. Cardiac asthma is usually nocturnal, waking the patient during the first hours of sleep. The attacks occasion great distress and anxiety, and there may be pallor, cyanosis, profuse sweating, and tachycardia, but recovery is the rule. ✓

Acute pulmonary oedema is another manifestation of left ventricular failure that may complicate hypertension. The patient is seized with sudden dyspnoea, a dry cough, and oppression in the chest, followed by copious expectoration of pink frothy albuminous fluid. Severe collapse may ensue.

Pulmonary embolism, if a large artery is blocked, causes sudden dyspnoea, cyanosis and collapse, with small rapid pulse. Pain is by no means constant at the onset, but follows with hæmoptysis when an infarct develops. Small emboli seem more often painful at the onset. The diagnosis must be made from the pre-existing condition of the patient, the likely causes of pulmonary embolism being a thrombosed vein, a recent surgical operation, or the presence of auricular fibrillation.

The immediate treatment of these three forms of acute cardiac dyspnoea should be an injection of morphia, preferably combined with atropine. In cardiac asthma and pulmonary oedema venesection always gives prompt relief, and 10–15 oz. of blood should be withdrawn. An intravenous injection of strophanthin, 1/100 grain, can be given if the patient's condition is grave. Once the attack is over, a period of rest in bed and a course of digitalis by mouth are required.

Palpitation.—The sudden onset of a rapid heart action, due to some form of paroxysmal tachycardia, may provoke faintness, dyspnoea, pain in the chest, or even syncope. If the heart is healthy little more than discomfort is likely to result, unless the attack is prolonged, but if the heart is diseased failure may rapidly develop. The attack can sometimes be arrested by the induction of vomiting or by firm pressure on the carotid sheath in the neck. If there is much distress, an injection of morphia is the best treatment. Signs of heart failure are an indication for digitalis or strophanthin. If the tachycardia is of ventricular type, as proved by an electrocardiogram, quinidine will usually stop it, and should be given, however serious the patient's condition. Five grains are given by mouth, and repeated hourly up to a total of thirty grains or until the attack ceases. Intravenous quinidine has been given in very urgent cases.

Special Points in Treatment.—In differentiating between cardiac and peripheral circulatory failure, a reliable history should be obtained as soon as possible. The relatives should be questioned as to the patient's state before the attack and as to any previous similar attacks. Syncope in the young is rarely serious, but in the elderly it is often due to cardiac or cerebral disease, and the prognosis should be guarded until a complete investigation has been made of the cardio-vascular and nervous systems, including a Wassermann reaction and an electrocardiogram.

The indiscriminate use of strychnine and digitalis in all forms of acute circulatory failure is to be condemned. Morphia is generally the best treatment for all forms of painful or dyspnoeic seizure. In cases of syncope, whatever the cause, adrenalin may give dramatic results, and should always be carried. Strychnine has no action on the heart, but large doses, e.g., 1/12 grain, are said to be effective in syncope or peripheral failure. The indications for digitalis are the presence of (congestive heart failure, with engorgement of the neck veins) or of auricular fibrillation or flutter. It is also of value in cardiac asthma. Digitalis has no effect in the peripheral type of circulatory failure. When given by mouth, adequate doses are essential, and a rapid effect may be obtained by a massive dose, for example, 10 grains of digitalis leaf or 2 drachms of tincture. Alternatively, strophanthin, 1/100 grain, or digoxin, ½ mg., can be injected intravenously.

CHAPTER III.

HEAD INJURIES IN CHILDREN

BY

CECIL P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S.E.

Senior Surgeon to King's College Hospital and to the West End Hospital for Nervous Diseases ; Consulting Surgeon to the Maudsley and Hammer-smith Hospitals.

A PROGRESSIVE increase, year by year, in the number of head injuries, a large proportion of which occur in children, is significant of the increasing danger to which life is subject nowadays. The prognosis in a case of head injury depends entirely on the extent to which the associated intracranial contents are damaged. Generally speaking the prognosis is more favourable in children than it is in adults, as the former have greater recuperative power than the latter. In very young children under two years of age the prognosis is usually unfavourable, as the intracranial venous sinuses, which are not well protected by bone at this age, are very liable to be damaged. From the age of two to puberty the prognosis is much more favourable ; although the head injury may cause a fracture of the vault, the fracture seldom extends into the base of the skull. Further, at this age there is much less likelihood of damage to or hæmorrhage from the meningeal vessels, as the inner table of the skull is not fully formed ; consequently these vessels are not enclosed at any part of their course in bony canals.

In children the membrane bones of the vault of the skull are very elastic, and for this reason trauma more often results in a bending rather than a breaking of the bone. In children the sutures of the skull are occupied by a considerable amount of cartilaginous and fibrous tissue, which serve as buffers, and thus tend to limit the effects of trauma. The dura mater is more closely adherent to the skull bones in children than it is in adults, with the result that any extradural hæmorrhage which may occur

is less extensive than in the latter. In children the skull bones contain very little diploë, and this anatomical fact prevents any difference in the extents to which inner and outer tables of the skull are fractured ; if one table is fractured, the other must necessarily be involved as well. The brain of a child is more tolerant of a traumatic lesion, and is thereby much less liable to permanent injury than is that of an adult.

Wherever possible, conservative measures should be adopted when treating head injuries in children. Compound or depressed fractures are, of course, exceptions to this dictum, and always demand operative interference. The child should be placed in a darkened room and treated for shock, but morphia must at all costs be avoided, as it acts as a depressant to the respiratory centre. Camphor in oil given hypodermically is the best stimulant for children. A careful record of the pulse, respiration and temperature should be kept. If a scalp wound exists, this should be carefully excised, and the underlying bone examined. If a depressed fracture be discovered, a small trephine hole should be made to one side of it, an elevator inserted, and the fragment elevated into position. No operation should be undertaken until the patient has quite recovered from the primary shock.

In many cases there is no evidence whatever of bony injury, but concussion is a marked feature, manifesting itself in children by giddiness, headache and vomiting. As there is practically always an increase in the intracranial pressure following a head injury, and this increased pressure is responsible for some of the phenomena of concussion, it is essential to reduce this in some way or other. Lumbar puncture lowers the intracranial pressure, and is a very useful form of treatment in these cases, but has the disadvantage that it disturbs the little patient very considerably, and it should only be used when other methods fail to relieve tension. Hyper-tonic salt solution can be given by rectum or intravenously, and has a marked effect in promoting the reabsorption of cerebro-spinal fluid, and thereby lowers the intracranial pressure. Magnesium sulphate may be given by mouth in milder cases of concussion, and produces similar results. In children with considerably increased intracranial pressure, 20 c.c. of a 10 per cent. solution of sodium chloride given intravenously will quickly relieve headache and irritability. A restless, irritable, incontinent child refusing food before the transfusion becomes a contented little patient

asking for food and comfort. In my opinion, lumbar puncture should be reserved for those cases in which hypertonic saline given intravenously does not reduce the intracranial pressure. Cerebro-spinal fluid should be withdrawn very slowly, and a control of the amount of fluid withdrawn by means of manometric reading is advisable.

In cases where the injury has produced a simple depressed fracture, no immediate operative treatment is necessary, but the shock and concussion should be adequately dealt with (Fig. 1). After an interval of two or three days, when all signs of shock and concussion have passed off, the depressed fracture should be elevated. That all depressed fractures in children should be operated upon within a week of the injury may be regarded as an axiom. If the operation be delayed for a longer period there is always a danger of epilepsy developing at a later date. The operative procedure consists in turning down a scalp flap sufficiently large to expose the fracture adequately. A small trephine opening is then made to one side of the depressed area. On removal of the trephine disc a fine dura mater separator is inserted between the bone and the dura mater. Generally speaking, this will be found to form quite an efficient elevator, and with it the surgeon can raise the depressed area to its normal position without any bleeding taking place. The dura mater should be carefully examined in order to discover any laceration or damage from which it may have suffered. The trephine disc *should not be replaced*, since there is a possibility of its undergoing displacement between the skull and the dura mater, and thus giving rise to pressure on the brain. The scalp should be closed with interrupted silk-worm gut sutures. It is unnecessary to take any precautions for drainage.

Compound fractures of the vault of the skull demand immediate treatment in order to obviate any possibility of infection. Delay in dealing with these fractures may result in meningitis, extradural or cerebral abscess, or even osteomyelitis of the cranial bones. The whole head must be shaved, and the scalp thoroughly cleansed. The wound edges should be completely excised, and any dirt or hair carefully removed. Should the fracture, when examined, prove to be a simple linear one, no further treatment is required. Here again the scalp is sutured with interrupted silk-worm gut sutures and no drainage is necessary.

In compound depressed fractures, however, the depressed

fragments must be elevated. If the underlying dura mater is torn a careful inspection of the underlying brain must be made, and any blood clot or debris removed. The dura mater need not be sutured, but is simply replaced over the cortex of the brain. A drainage tube should be inserted down to, but not through, the hole in the dura mater, and is to be removed after an interval of forty-eight hours. In all compound fractures of the skull anti-tetanic serum should be administered. Urotropin should be given in doses of five grains every four hours for several days following the operation. If this drug is given by mouth, it makes its appearance in the cerebro-spinal fluid in the form of formaldehyde, and, as such, helps to counteract any threatened infection of the meninges.

Basal fractures of the skull rarely require any operative interference, unless there are signs of intracranial hæmorrhage or œdema. In such cases a decompression operation is necessary. In many cases a fracture of the base in a child is fatal, and death instantaneous (Fig. 2). As a rule the posterior and middle fossæ are the parts of the skull which are chiefly affected.

An X-ray examination of the skull in children may be a valuable aid in confirming the diagnosis of a fracture. On the other hand, it may prove useless, especially in cases where the fracture is basal and passes through the petrous portion of the temporal bone.

Although no operative interference is called for in the majority of basal fractures, a very definite line of treatment should be followed in these cases. Careful attention must be paid to those cavities which communicate with the base of the skull. All blood clots should be carefully removed from the nose with small pledgets of sterile absorbent wool. The same treatment should be repeated with the external auditory meatuses. The practitioner may be worried when a definite fracture of the base of the skull occurs in a child in which a chronic aural discharge is present. In such cases no syringing of the auditory meatus whatever should be allowed, as the constant discharge of cerebro-spinal fluid through the fracture of the petrous temporal bone into the middle ear, and thence into the external auditory meatus, may serve as a preventive to the occurrence of meningitis. On the other hand, syringing in such cases with an antiseptic may actually bring about this dreaded complication.



FIG. 1.—Photograph of child showing a large depressed fracture in the left frontal region. This was caused by a fall from a window. The child was operated on four days after the injury, and the fracture was elevated. The child made a complete recovery.

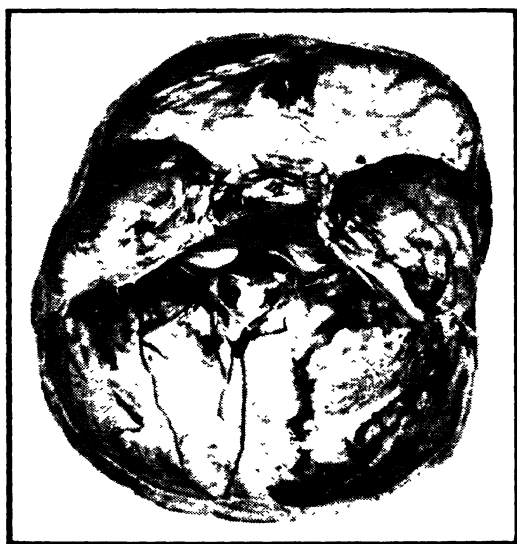


FIG. 2.—Fracture of the base of the skull involving the posterior fossa and middle fossa in a child aged five. A post-mortem specimen.

'HEAD INJURIES IN CHILDREN,' by Cecil P. G. Wakeley, D Sc.
F R C S, F R S E.

In order to avoid subsequent trouble, the most important part of the after treatment of head injuries is prolonged rest, and this is often the most difficult part to enforce. The little patient often feels quite fit after a few days, and implores to be allowed to get up and play. However, three weeks' rest is the absolute minimum after any severe head injury. Parents themselves often take the part of the child, and are usually anxious that school should be recommenced at much too early a period. To be on the safe side the practitioner should insist upon prolonged rest for a month or six weeks at least. It is only by these means that remote complications in the form of epilepsy can be avoided.

CHAPTER IV.

THE MODERN TREATMENT OF BURNS AND SCALDS

BY

PHILIP MITCHINER, M.D., M.S. Lond., F.R.C.S. Eng.

Hon. Surgeon to H.M. The King; Surgeon with charge of the Septic Wards
and Senior Surgeon to Out-patients, St. Thomas's Hospital, London.

It is, of course, in the treatment of burns and scalds that tannic acid has its greatest use, both as a first-aid dressing and for subsequent routine treatment; and it has been found at St. Thomas's Hospital that a solution of more than 2 per cent. tannic acid has no advantage over greater strengths when used either as a compress or for spraying.

If we consider the severe collapse met with in burns and scalds we must realise that tannic acid is the ideal dressing, applied preferably in compress form, as this is applicable to private practice, at the patient's home, or in the hospital out-patients' department, where the spray method is impracticable. This collapse is due partly to the fright and upset of the accident, but much more largely to the absorption of protein bodies from the damaged tissues and the great loss of fluids which occurs from the burnt area; and the tannic acid, by completely coagulating all these damaged tissues, prevents these latter causes of collapse: it is obvious, therefore, that the earlier the tannic acid is applied, the less collapse will result from the burn. For the shock due to the fright, no drug is better than opium or its derivative, morphia, and this should be administered early and freely, preferably in the form of tinct. opii.

First-Aid Treatment.

For convenience in works and other places where burns are likely to occur, tannic acid can be made up into a tablet with 1/2000 perchloride of mercury, to act as a mild antiseptic and enable the solution to be made up safely with warm tap-water. No other

antiseptic has proved so satisfactory and painless as the perchloride of mercury, though I have tried many at St. Thomas's Hospital.

R

Tannic acid	gr. 17½
Perchloride of mercury	gr. 7½
Boric acid	gr. 1

This tablet keeps indefinitely ; it should be crushed well before adding the water, as otherwise solution is slow. The tablet is dissolved in 2 oz. of warm water, and gives sufficient 2 per cent. solution to soak a dressing of lint 8 in. by 4 in.

The lint should be clapped on quite wet to the burn, and the patient should be left quiet for at least twenty minutes before being moved to an institution for further treatment, being in the meanwhile, of course, kept warm and given fluids to drink ; while,

TABLE.

OPIUM DOSAGE FOR CLEANSING BURNS AND SCALDS.

I.—IN CHILDREN.

Age.		Preparation.	Dose.
1/12	...	Tinct. camph. co.	min. ii-iii
2/12	...	" "	min. iv-vi
3/12	...	Tinct. opii.	min. ½-¾
6/12	...	" "	min. ¾-1
1 year	...	Tinct. opii (a)	min. ii-iii
		or	
Over 1 year	...	Inj. morph.	gr. 1/75
		Tinct. opii	min. ii for each year
		or	and min. ii in 15 mins. if necessary
		Inj. morph.	gr. 1/75 for each year

II.—IN ADULTS.

12-15 years	...	Tinct. opii	min. xxx
		and	
		Inj. morph.	gr. ½
15-20 "	...	Tinct. opii	min. xxx
		and	
		Inj. morph.	gr. ½
Over 20 years	...	Tinct. opii	min. xxx
		and	
		Inj. morph. (b)	gr. ¼-½
		or	
		Inj. morph. (c)	gr. ½-1

Remarks.—(a) Tinct. opii is more satisfactory than morphia. (b) For women. (c) For men according to stamina.

N.B.—Should slow or shallow breathing give rise to anxiety, atropine sulphate, gr. 1/200-1/50 should be administered hypodermically.

if a medical man is present, morphia or tinct. opii should be administered in dosage according to the Table, after which the patient must be left undisturbed until the drug has taken full effect (usually about twenty minutes) before proceeding with the cleaning which is a necessary preliminary to the final dressing.

Routine Treatment.

It is essential that this first-aid dressing, even if of tannic acid, should be removed, which it can be most easily, and the area carefully cleaned before the final tannic acid dressing is applied. By this careful cleansing alone can subsequent sepsis be avoided ; and here let me say that should sepsis supervene during a course of treatment, it is only necessary to remove the tannic acid dressing, re-clean the area thoroughly, and re-apply a new tannic-acid dressing in order to get a good result.

The cleansing must start with the removal of all dead and charred tissues, the excision of all loose skin raised over blisters, and the removal of grease, especial attention being given to the edges of the damaged area. The whole area should then be gently but thoroughly cleansed with soap and water (applied with a sterile swab and *not* with a nailbrush), and finally carefully sponged over with ether, to remove all natural or applied grease, in order to get a thorough coagulation. It is obvious that this treatment cannot be carried out unless the patient is rendered insensible to the pain which such a cleansing of the damaged area must entail. We have found at St. Thomas's that it is safer and more satisfactory to procure this insensibility by the administration of large doses of opium rather than the use of general anæsthesia, which tends, even with the greatest care in its administration, to be followed by pulmonary complications, usually of a mild nature, which are detrimental to the rapid recovery of the patient. The Table indicates the dosage and drug employed at various ages, and has proved both safe and satisfactory.

A compress consisting of either six layers of sterile gauze or three layers of lint should be thoroughly soaked in a stock solution of 2 per cent. tannic acid and 1/2000 perchloride of mercury, and applied closely and evenly over the entire burnt area without wringing the dressing out. This compress should then be firmly and evenly bandaged in position, the bandage being applied directly to the outer side of the compress. The whole of the outside of the

bandage may then be soaked with a spray of 2 per cent. tannic acid and 1/2000 per chloride of mercury in order to ensure that the underlying dressing is thoroughly wet. Mackintosh or jaconet should be placed under the patient until the dressing has dried, in order to save soiling of the sheets by the tannic-acid solution ; for though this all washes out excepting for a faint iron-mould, the sheet subsequently rots in this area. It is also well to secure the burnt limb temporarily in order to prevent undue movement until the tannic coagulum has formed firmly. This dressing should be left in position and untouched for at least a fortnight in the case of small burns and scalds, and three weeks where large areas are involved. At the end of this time the bandage should be cut and the dressing lifted, when the scab usually will separate completely from the burnt area. If it does not do so the dressing can be re-bandaged and removed later when separation has occurred. The separation may be rendered quite painless, and assisted by spraying with tannic-acid solution between the coagulum and the skin during this process. May I here say that the escape of a little serous or sanio-serous fluid from the edges of the coagulum, or the raising in blisters of its centre with serum, is no more an indication for interference or removal of the dressings during the first week after their application than it is with the bare coagulum resulting from the " spray " method.

Temperature and toxæmia as shown by a rapid pulse, dried and furred tongue and pain, with possibly the escape of sero-pus in considerable quantities, are the only indications calling for removal of these dressings before the time stated, and these indications in our experience are extremely rare, and usually result from a failure in the thoroughness of the preliminary cleansing. Should removal be necessary, and in my experience it is very seldom called for, the area should be re-cleaned carefully, and another tannic-acid dressing applied.

As indicated, the dressing must be closely and evenly applied to the burnt area ; and there are certain areas, therefore, such as the perineum, the groin, axillæ, the neck, and also the face, where the absence of bandages is an advantage, and where the spraying method gives better results ; but the compress method has been, and can be, used with good effect in these areas.

It should be borne in mind that in burns where destruction of the subcutaneous and muscular tissues has occurred, subsequent

scarring and contracture are inevitable, and it is, therefore, necessary to apply tannic-acid dressings, either by compresses or spraying, with the part in such a position that subsequent contracture may be counteracted. Equally is it obvious that although full epithelialisation will have occurred in those areas where the first, second or third degree burn has occurred, an ulcer must be expected, and will be met with, in deeper burns; and this, if extensive, should be treated by subsequent autogenous skin grafting, or, if small, by the application of some lotion, of which, in my experience, the most satisfactory is the following: Purified alum, gr. 20; zinc sulphate, gr. 10; glycerin, 7 fl. oz.; water, to one pint.

Mortality.

It must be realised that the mortality from burns is very much greater (approximately three to one) than from scalds, where the hot fluid is only in contact with the tissues for a few seconds; and a more guarded prognosis therefore must be given in the case of burns.

It has been shown that whereas with burns treated with oils, etc., death usually occurs from secondary sepsis rather than immediate collapse, both these causes are largely eliminated in the tannic-acid treatment, which again is more efficient than picric acid, where only the surface of the damaged tissue is coagulated, and so collapse from absorption in the deeper parts is not prevented, though sepsis is largely eliminated. The attached mortality table speaks eloquently for the value of tannic acid:

MORTALITY OVER FIVE-YEAR PERIODS.

Dressing.	Burns.	Scalds.
Oil, etc. ...	39.0%	18.8%
Picric 1% ...	15.5%	7.0%
Tannic 2% ...	4.0%	1.7%

Other Uses of Tannic Acid.

(1) In chemical burns the tannic-acid treatment is also applicable and very successful, but the burning agent must be neutralised before applying the tannic acid. (2) In any superficial wound, such as grazes or abrasions, or the areas from which skin grafts have been removed, or where extensive curettage has been employed, as in dealing with lupus, the tannic-acid compress makes an excellent

and quite painless dressing ; and on its removal at the end of approximately three weeks the area will be found completely epithelialised. (3) Another use for the tannic-acid compress is in sunburn, where the results are excellent and the immediate relief obtained most satisfactory. Unless actual blistering is present it is usually sufficient to retain the compress for two or three days only in these cases. (4) A further use can be found in the treatment of radium and X-ray burns.

It will be seen from the foregoing remarks that the tannic-acid compress has many uses apart from the treatment of burns and scalds, where it has been so successful in removing both the sepsis and reducing the mortality, and it is therefore to be hoped that its uses may become more widely known and the practical method of compress popularised.

CHAPTER V.

OBESITY IN WOMEN

BY

ETHEL BROWNING, M.D.

Assistant Pathologist, Pickett-Thomson Research Laboratory, St. Paul's Hospital.

OBESITY in women has been regarded of late years chiefly from the æsthetic point of view. The dictates of fashion have set up a standard of slenderness, sometimes in an exaggerated form, to which the majority of women have thought themselves bound to attempt to conform. Many, however, who were decidedly overweight before they made their æsthetic effort, have found themselves to have attained not only the desired improvement in appearance, but also a physical and physiological efficiency unknown to them during their years of obesity. For obesity undoubtedly involves not only discomfort, but very real danger to health. It is well known that the mortality rate of the obese is far higher than the average, and that their risk from operation, pneumonia or infectious disease is very high. Obesity shows also a frequent association with diabetes and gall-stones; Joslin found marked obesity as a precursor of diabetes in 40 per cent. of 1,063 cases.

Circulatory deficiency is an almost invariable concomitant of pronounced obesity. The deposits of fat frequently found around and in the heart muscle not only constitute a mechanical disadvantage, but may also cause physico-chemical disturbances in the cell metabolism. These disturbances are shown by abnormalities of the blood pressure (usually hypertension) and of the pulse rate, by diminished capacity for work and by electro-cardiographic changes (Master and Oppenheimer, 1929).

Factors in the Preservation of Normal Body Weight.

The preservation of the normal body weight depends upon :

(1) *The Food Intake*.—An amount of food taken in excess of the metabolic requirement is stored as fat in the tissues. The number of calories needed to maintain the body weight can be

calculated from the approximate standard of thirty calories per kilogram, so that an individual weighing ten stones requires two thousand one hundred calories daily. If ten stones is greater than the ideal weight for the individual height and build, it follows that the intake of two thousand one hundred calories or more is too high for the maintenance of weight without depositing fat, and if such an intake is long continued, marked obesity will eventually result.

(2) *The Individual Basal Metabolism.*—In simple obesity, uncomplicated by endocrine deficiency, the basal metabolism is usually normal. It has been shown, however, that in some obese persons what is known as "luxus consumption" occurs, *i.e.*, an increase in basal metabolism may take place as an adjustment to constant overfeeding, as was demonstrated in dogs by Grafe and Graham (1912), and Eckstein and Grafe (1919). A similar adjustment may take place when, as sometimes happens, the resistant point to a fixed reducing weight is reached, and the weight remains stationary, or even begins to rise again. In such a case the obese subject has adjusted her metabolic requirements to her reduced intake.

(3) *The Specific Dynamic Action of Foodstuffs.*—The effect of foodstuffs in speeding up combustion has been found in some cases to be not so marked in the obese as in the normal individual. According to some observers—Wang and Strouse (1924), Gibbons (1924), and others—there is in obese patients not only a diminished specific dynamic action of protein, but also a capacity for using protein and carbohydrate more easily than fat, so that as long as any protein and carbohydrate is available no fat will be used.

(4) *Exercise.*—Increased combustion of the food intake supplies the energy necessary for muscular exercise. It is often impossible for the obese woman to take exercise sufficient to influence food combustion in any significant degree, owing to the long strain on the circulatory organs and the lack of tone of the skeletal muscles. The mild exercise, which is all that is possible, often has only the effect of stimulating the appetite and leading to a greater intake of food.

Types of Obesity.

It has long been customary to divide cases of obesity into two classes: the simple or exogenous, due to overeating and muscular inactivity; and the endogenous, due to some defect in metabolism

or to endocrine deficiency. It is now recognised, however, that there is no strict dividing line between these two types. The majority of cases are a mixture of these two and of a third—the “constitutional” type.

The part played by heredity in “constitutional” obesity is not completely agreed upon. In 523 cases examined by Dunlop and Murray Lyon (1931), 69.2 had parents who suffered from obesity, the maternal heredity being more common than the paternal. It has often been pointed out that it is difficult to distinguish between the influence of heredity and early environment in obesity, the children of parents with a tendency to overeat being led in the path of overfeeding from childhood. That the endogenous type of obesity is more common in women than the exogenous, has been said to be indicated by the fact that sudden increases of weight are commonly observed at puberty, after childbirth, and at the menopause.

It has been very well shown by Evans and Strang (1929), however, that the so-called endocrine obesity can be treated with success by dietary measures alone. As an example of how exogenous obesity may bring endogenous obesity in its train, the theory of Falta (1925) with regard to hyperinsulinism may be mentioned. Falta believes that in obesity there is a tendency to heightened activity of insulin, which is known to promote assimilation, in contradistinction to the action of thyroxin. According to Falta, a constant excessive carbohydrate intake may over-stimulate the pancreas, the hyper-insulinism thus produced further increasing tissue assimilation, so that a vicious circle is induced.

Among the other endocrine glands, the thyroid and the ovaries have been chiefly incriminated as the cause of female obesity. There is no doubt that many cases showing a lowered metabolism show rapid loss of weight when thyroid treatment is added to a fixed reducing diet. Treatment with ovarian hormone has given variable results; success having been reported in some cases of menopausal obesity. Cases of adrenal and pituitary obesity are usually definitely and pathologically recognisable, and scarcely come within the scope of this article. Nor do the cases following lesions of the central nervous system, such as encephalitis lethargica or neoplasms in the region of the hypothalamus. It is interesting to note, however, that some observers, including Grafe and Grunthal (1929), and Bernhardt (1930), have postulated the existence of a

metabolic centre controlling normal weight in the hypothalamic region, and that Bernhardt believes this centre can be influenced by peripheral disturbances, in which he includes endocrine deficiencies.

The Treatment of Obesity.

Whether the case is one of simple exogenous obesity, or whether, as most commonly occurs in women, it is complicated by an endocrine deficiency not attributable to a pathological condition of any one gland, the essential element of treatment is dietary restriction. Whatever element—protein, carbohydrate or fat—bears the brunt of the restriction, the total calorie value must be lowered.

The amount of the reduction varies according to the weight and general condition of the patient, but in practice a daily intake of 900–1,000 calories is usually found effective in producing a gradual loss of weight without great discomfort or disturbance of health. Advocates of special diets differ in the distribution of the allowed calories between the carbohydrates, proteins and fats: the Salisbury diet, for example, recommends 380 gm. of protein; while Evans and Strang (1929) report excellent results from giving the very minimum of fat. A diet giving a good average of success will contain 40–120 gm. of carbohydrate, 50–180 gm. of protein, and 25–120 gm. of fat.

Other Factors in Successful Treatment.

(1) *Preservation of the Nitrogen Balance.*—If protein is cut down too drastically, wastage of the tissues may occur. In order to avoid this the proportion of protein to carbohydrate should be approximately 1 gm. of protein to 0.6 gm. of carbohydrate, and the total amount of protein not less than 60 gm.

(2) *Minimum Fat Intake.*—If the intake of fat is cut down to a minimum the stored fat will be called upon to supply its metabolic place, and the loss of weight will be more rapid than when a moderate amount of fat is taken. According to Evans and Strang (1929) the possibility of the development of acidosis on such a diet is not great.

(3) *Avoidance of Hunger.*—In order to avoid the discomfort involved in giving small concentrated meals, they can be made bulky by giving a high proportion of fruit and vegetables which have low calorie value.

(4) *Maintenance of Water Balance.*—It is not uncommon for obese patients who lose a good deal of fat to store water in the tissues, so that the weight becomes stationary or even rises. This can be prevented by giving an almost salt-free diet, and by increasing the amount of protein.

(5) *An Adequate Mineral and Vitamin Content.*—Since a satisfactory reducing diet should contain plenty of fresh foods (fruit, salads, etc.), the mineral intake and that of vitamin C is in no danger of deficiency. If the fats have been very much reduced there is some likelihood of vitamins A and D being reduced below the optimum level. In such a case concentrated preparations of vitamin A (carotene) and vitamin D (irradiated ergosterol) can be given. If a special form of starch-reduced bread is given, and other cereals are severely restricted, there is a possibility that the vitamin B content may be inadequate. This can be obviated by giving a cup of yeast extract once a day instead of clear soup.

(6) *The Psychological Factor.*—The effort of will required, especially where long habit has accustomed the patient to a diet not only too great in quantity, but consisting of rich and stimulating foods, to change to a diet of small quantity and simple character is often considerable. It may be made easier by a suitable choice of foods, and their distribution over the various meals allowed. In the majority of cases a slow reduction is preferable, from the point of view of comfort and maintenance of strength, to a very rapid one, and if it is explained to the patient that the reduction, though slow, will be steady under such a régime, she is more likely to persevere than if she expects a rapid loss which fails to take place.

The following is a record of an actual typical case of moderate obesity which responded very well to a considerable restriction of diet, without any discomfort, and with a marked improvement in general health.

A woman, aged fifty-four, 5 ft. 6 in. in height, weighed 11st. 4 oz. She stated that her normal weight was about 9 st., and that the extra weight, which had gradually accumulated during the past three or four years, was a source of discomfort and fatigue. She had no obvious signs of thyroid insufficiency, therefore it was considered unnecessary to give thyroid extract. Her calorie intake was restricted to about 900 calories daily. The following diet was prescribed :

Breakfast : Tea or coffee with milk, but no sugar ; starch-reduced bread (one roll=seventy calories) ; one pat of butter ; grape fruit, orange or apple. 11 a.m. : Cup of marmite, or clear soup, or beef essence. Lunch : One egg, or piece of cheese, or steamed fish ; one roll with one pat of butter, and green salad with tomato and dressing of olive oil and vinegar, or green vegetables ; any raw fruit, or stewed fruit without much sugar. Tea : Tea with milk, but no sugar ; dry toast or biscuits, one pat of butter. Dinner : Clear soup ; lamb cutlet or lean steak or veal (3 oz.), grilled, or white fish steamed or grilled, or chicken ; green vegetables (two, if liked) ; no potatoes ; fresh fruit salad, baked apple, etc. ; small piece of cheese, one roll or dry biscuits. At bed time : Glass of hot water with juice of one lemon.

Since the patient complained of muscular and joint pains, accompanied by fibrositic nodules in the painful muscles, she was advised to have massage twice a week ; and since she also complained of " hot flushes," she was given injections of ovarian hormone. Under this régime she lost weight at the rate of one and a-half pounds to two pounds a week, until at end of three months she had regained her ideal weight of nine stones. By this time also the fibrositis had disappeared, and she described herself as " perfectly fit and well," and stated that the diet caused no inconvenience or discomfort. Her weight has remained stationary at nine stones for the last eighteen months, and her general condition entirely satisfactory.

The results of treatment in this case confirm the opinion held by Evans and Strang, that " no person, whatever his endocrine function may be, will gain in weight if his caloric intake balances or is less than his energy output. And except in those rare cases in which obesity is associated with the true endocrine disorders, too widely known to need description here, in which endocrine therapy should be instituted for the endocrinopathy itself, endocrine products should not be thought of in connection with obesity. The treatment should be dietary, and dietary alone."

CHAPTER VI.

OBESITY IN MEN

BY

IVO GEIKIE COBB, M.D., M.R.C.S.

Physician, Norwich Union Insurance Company; Consulting Endocrinologist,
Besford Court Mental Hospital.

Introductory.

IF we accept the definition of obesity as an excessive accumulation of fat in the body, it is necessary to define as near as possible the word "excessive." It will be obvious that the normal quantity of fat which is present in a man merges imperceptibly into the condition known as adiposity. For practical purposes we may define obesity as an increase of 20 per cent. of fat over that found in an individual of average weight and height. Obesity, therefore, may be reckoned as a condition in which fat accumulates in the body so that the individual's weight is 20 per cent. over that normal for his age and height. It is customary to divide obesity into exogenous and endogenous.

The exogenous variety is that which arises from causes outside the bodily economy. In other words, the condition arises as the result of an excess of intake over output. The endogenous variety consists of an altered function of one or more of the organs concerned in the regulation of metabolism. We are here concerned with obesity in men, and it will probably be suitable if we consider our subject, first, from the standpoint of juvenile obesity, and, secondly, obesity in the adult.

Exogenous Obesity.

Owing to several factors which are active during childhood and adolescence, it is probable that cases of exogenous obesity during these epochs are rare. There is, of course, in the ordinary child an activity which, for practical purposes, precludes a deposit of fat due solely to overeating. While it is obvious that many children overeat, more especially at such festive times as Christmas, the result is nearly always shown rather by the expulsion of food

by an indignant stomach than by its retention as excessive fat. It is a well-recognised fact that fat is not deposited on muscles which are exercised, and even in children with comparatively obese trunks the extremities are rarely equally fat.

When we come to consider exogenous obesity in the adult, we are faced with the lamentable conclusion that it is the result, in a very large proportion of cases, of overeating. It is true, of course, that some individuals are punished for their over-indulgence at the table by the unlovely accumulation of fat ; while others, equally self-indulgent, escape the penalty which should be theirs. This, of course, is due to the fact that the latter group of persons are blessed with an active catabolic mechanism.

As youth merges into middle age, violent exercise is replaced imperceptibly by exertion of a less drastic nature. The young man gives up football for tennis, and this in turn is replaced by golf, gardening or walks. How rare it is, however, for a reduction either in the quantity or quality of the nourishment ; although common sense would suggest that when the utilisation of foodstuffs is no longer so large, the allowance should *pari passu* be curtailed. That this fact is not recognised is borne out by the common but unpleasant sight of middle-aged men whose bulk has increased out of all proportion in the decades twenty-five to forty-five. The insurance companies allow an increase of a stone in weight during these years, but for this there is really no excuse. It has rather come to pass because of the frequency with which it is encountered. It is probably also true that the muscles of the trunk—which have been amply exercised by football and rowing—sink into disuse once these strenuous exercises are abandoned.

This form of obesity calls for drastic criticism on the part of the doctor. It is an act of extreme folly to allow the important viscera of the thorax and abdomen to become plastered with layers of fat. The protuberant abdomen contains pounds of excessive adiposity hidden in the omentum and mesentery, while every medical man will have noticed how the fatty tissue is deposited in such localities as the pericardium and abdominal muscles. When such a deposition of fat is found it is important to decide whether the case falls into the class of exogenous obesity, or whether the unfortunate sufferer is in reality a case of endogenous obesity. If the former, provided heart and circulation are so far unimpaired, a radical alteration in the diet and amount of exercise is essential.

A course of physical exercises directed to the abdomen, buttocks and hips should be laid down firmly for the patient. It is hardly necessary to mention perhaps that these should not be of too severe a character to commence with, but should gradually be increased as the patient becomes more supple and therefore the exertion of moving becomes less.

Endogenous Obesity.

Fortunately for the reputation of the diagnostician, the main types of endogenous obesity stand out fairly clearly. As these are entirely concerned with the endocrine organs it will be easier to study them here under their various headings.

Obesity of Thyroid Origin.—The picture of thyroid obesity is clear cut. It should be mentioned at this place, however, that many cases of endogenous obesity are not purely thyroid in origin, although this important gland is concerned somewhere in the picture. It is, however, admitted that thyrogenous obesity tends to run in families. It may be noticed first of all after an infectious disease, in which case there is obviously some toxic basis. The congenital factor, however, is shown by the fact that, while many people suffer from infectious diseases, only a certain proportion subsequently develop adiposity. The distribution of the fat is characteristic in that it is almost entirely confined to the trunk. There is the “boss” of fat over the *vertebra prominens*, which gives rise to the well-known “Cassowary neck.”

It is possible that the infectious disease which precipitates the condition has set up some degree of thyroiditis. If the basal metabolism is examined it will probably be found to be markedly under the normal limit, although there are cases in which it is but little reduced. Almost it seems as if these subjects are further cursed with a large appetite, although in nine cases out of ten this they will indignantly deny. It is also unfortunate that a reduction of diet by itself helps but little, although the exhibition of thyroid substance literally places the right key to the right lock.

Hypophyseal Obesity.—The best known syndrome of hypophyseal obesity is that associated with the name of Fröhlich. This condition, which is also known as dystrophia adiposogenitalis, is a well-recognised form of juvenile obesity. Apart from such organic causes as cerebral tumours causing pressure on the pituitary gland, or internal hydrocephalus (which may lead to the state

known as cerebral adiposity), the cases to be considered under this heading are mainly those due to functional disturbances of the pituitary gland. This form of obesity is very characteristic in its distribution. The fat is deposited mainly on the breasts, abdomen, hips, buttocks and suprapubic areas. There is also a tendency to a feminine type of pelvis with knock-knees, and a non-masculine type of pubic hair. If the cause occurs in childhood, there is a lack of development of primary and secondary sexual characteristics. A radiogram of the *sella turcica* may show some abnormality either in the direction of enlargement of this bony area, or some such deformity as fusion of the clinoid processes.

There are certain symptoms associated with this state. In the female there is amenorrhœa, or at any rate scanty periods. In both sexes there is lack of concentration, drowsiness and often backwardness. The children are, however, often original in their mentalities, and I have frequently been struck with the fact that children of this type cannot do mathematics. When this state is recognised early enough in life, the greatest care is needed throughout the whole of adolescence. In the slighter cases it is often said that the condition will right itself some time in the near future ; and the pious hope is expressed that puberty will adjust the faulty endocrine balance. It cannot be too strongly emphasised that such faith should be strongly supported by medical treatment, both dietetic and endocrine. Even if recovery is achieved without medical help it will at best lead to the loss of a year or two's maturity, and at worst will handicap the child to such an extent that he may never catch up with his fellow men.

Obesity and the Genital Organs.—Much work has recently been done on the relation of the pituitary gland to the gonads. The discovery that the pituitary gland secretes an œstrin which is essential to the functions presided over by the ovaries, has thrown light upon many hitherto disconnected observations as to the rôle played by the pituitary gland. Many years ago, Tandler and Grosz showed that castration in the male produced the same fat distribution and the same type of pubic hair as is seen in hypo-pituitarism. The failure of the epiphyses to unite, and overgrowth of the bones of the extremities is seen, and, more important still, secondary enlargement of the pituitary occurs. The inter-relation of the pituitary and the testes is therefore apparent ; the pituitary appears to be held in check by the presence of the interstitial cells of Leydig,

and their potent hormone. The removal of this break—certainly before growth has ceased—allows the pituitary to function, no longer hampered by the gonads.

There can be little doubt, moreover, that during the middle years of life man undergoes a climacteric, less sudden, less sensational and less obvious than that seen in his feminine counterpart. It is at least possible that the ease with which obesity is achieved during middle life in men is partly due to a general lowering of the activity of the catabolic glands. The male climacteric, while not so definite as the female, is none the less suggested as a real factor by such changes as are seen in the prostate, which tends to hypertrophy; by the testes, which tend to atrophy; and by the analogous changes in other endocrine organs, no doubt brought about by what has been called hypo-endocrinism. This would naturally be accompanied by an increase in the fatty tissue of the body. The lowering of the basal metabolic rate, such well-recognised changes as a sub-normal temperature, a slow pulse, and subjective feelings of chilliness, all suggest that the thyroid, pituitary and gonads require some help in their uphill task.

A few words about the metabolism in obesity: It has been shown that overeating may increase the basal metabolic rate. From this it has been argued that heavy eaters have a higher metabolic rate than light ones, and that in obesity "this assumed normal response to overfeeding may be lacking."* In certain types of obesity it appears that there is a retention of water in the tissues. The nitrogenous metabolism is normal even during reduction cures unless these are extremely drastic, in which case there is a marked increase in the loss of nitrogen. Normally, however, there is an increase in the combustion of body fat, although protein is spared. In thyrogenous obesity there is a well-marked reduction of basal metabolism. The same thing has been noted, but to a less extent, in the obesity of hypopituitarism. It is well known that there is increased sugar tolerance in these cases, and that subjectively the subjects of these conditions frequently show a marked liking for carbohydrates.

Treatment.

Finally, a few words as to treatment. Every case of obesity, no matter whether endogenous or exogenous, requires a diet to some extent or other. A strict limitation of carbohydrates and

* "Endocrinology and Metabolism," Vol. iv, p. 42.

fat, but an insistence on adequate proteins, forms the basis of the diet. It is a disputed fact whether the restriction of the intake of fluids is beneficial. Many cures of obesity include restriction of fluids, and while weight is lost, due to loss of water, the restriction appears to have little effect on metabolism *per se*. Although there are many devices to assist the hungry man to restrain his appetite, many of these are more amusing than practical. It is a fact that less food is eaten if the meal is taken dry, but the plan of many and frequent small meals (designed to prevent hunger), while reducing the total intake, does not appear to have very much to recommend it. It has been suggested that the order of a meal should be reversed, and that instead of tickling the palate by soup (so as to make its owner deal more vigorously with the subsequent courses), the first course should be dessert, which should somewhat effectively interfere with the deglutition of the subsequent dishes. The most important facts are diet and exercise, with whatever alteration in the habits of eating which appears to be suggested by the individual case.

With regard to other treatment: The outlets from the body should be watched. The bowels, kidneys, skin and lungs should be encouraged to deal with the waste products which reach them. That this fact is well recognised is shown by the composition of many fat cures on the market. These will be found to consist largely of aperients and diuretics.

Thyroid therapy is invaluable, not only in thyrogenous obesity, but in other forms in which metabolic stimulation is required. Treatment by means of pituitary extracts in their various forms has become of increasing use as the hormones of this vital organ have been sorted out from their complexities. Many clinicians incorporate extracts of the gonads in one form or another, but opinion is still divided as to their utility.

CHAPTER VII.

THE MODERN TREATMENT OF EARLY SYPHILIS

BY

M. SYDNEY THOMSON, M.A., M.D., F.R.C.P., F.R.S.E.

Physician for Diseases of the Skin to King's College Hospital
and the Belgrave Hospital for Children.

SYPHILIS being so polymorphic and widespread in its effects, it is impossible to attempt a comprehensive survey of its treatment in brief. The later stages in which, *e.g.*, the nervous system is involved all exhibit their particular problems. But the effects of old-standing syphilis can only be ameliorated, whereas there is evidence that the disease can be cured if efficient treatment is introduced during the primary or secondary stages. From the point of view of the public health this fact is of the greatest importance, and for that reason only such treatment will be dealt with here.

“Modern” methods may be interpreted as dating in the first place from the introduction of the organic arsenical compounds, and particularly from the discovery of the neutral or “914” group. These latter have rendered intravenous therapy comparatively simple, yet their possible toxicity is such that certain preliminary precautions must always be taken. Disease of the kidneys, liver or heart necessitates lower dosage, closer observation, or even in severe cases total exclusion of “arsenic.” The excretion of too much “arsenic” through a previously damaged kidney can obviously aggravate that condition. The liver is the temporary storehouse of the drug, whither it is carried and retained for a time as a fat compound, the danger of acute degeneration of that organ being thereby explained. In long-standing cases it is always possible that the cardiac condition, whether valvular disease or a myocarditis, may either be primarily syphilitic, or may have been secondarily invaded by the spirochæte. For among all

affections syphilis is the supreme example in which a "locus minoris resistentiæ" is attacked. Should there be present some endarteritis of a coronary vessel there is danger of its total occlusion as a result of the Jarisch-Herxheimer reaction, *i.e.*, the injection of "arsenic" causes the death of spirochætes and the release of their toxins; these in turn excite a local inflammatory reaction with local increased pressure sufficient to obliterate the lumen of the artery.

In considering these possible dangers, it is obvious that the risks increase with the age of the patient as a result not only of the increased possibility of disease, but also of arterio-sclerosis. But although these dangers are real and cannot be disregarded, reasonable care will prevent accidents. More uncommon difficulties are sometimes encountered, *e.g.*, in a patient with widespread eczema a generalised dermatitis may be excited. On the other hand, the common "nitritoid" crisis which occurs immediately after injection has no pathological significance.

Bismuth has undoubtedly proved itself a very valuable drug, but whilst many authorities maintain that it has completely usurped the place of mercury, others feel that it is still premature to make so definite a claim. Nevertheless, it is true that bismuth is used to the total exclusion of mercury in some clinics. But whilst we are dealing in the case of "arsenic" with only two compounds, "606" and "914," under different proprietary names, the choice of a bismuth compound is more difficult. The following brief considerations, however, will indicate the lines along which it is used. If "arsenic" is being injected actively, it is better for the bismuth to be more slowly absorbed. An insoluble compound such as the salicylate suspended in oil is then given intramuscularly. If "arsenic" is in active use, but it is yet desirable to have the bismuth effects produced more rapidly, an oil-soluble salt such as the campho-carbonate is injected. Where "arsenic" cannot be given, a water-soluble preparation such as thiobismol or the potassium bismuth tartarate is used on account of its rapid absorption. Whatever the compound used, the only complication of practical importance is the characteristic "green line" stomatitis, which is usually of some severity.

A consideration of the following courses of treatment will reveal the diversity of opinion that still exists on this subject. Each one is designed to produce a cure in a case of primary syphilis

in which the Wassermann is still negative, the clinical diagnosis having been confirmed by the discovery of *Spirochaeta pallida* in the lesion. In each course it is assumed that progress is uninterrupted by absence or relapse. Harrison recommends weekly injections of "arsenic," the first three of 0.45 gram, the next two of 0.6 gram. At the same time weekly injections of a bismuth preparation are given. For the next three weeks ten grains of potassium iodide are given t.d.s. by mouth. Both the bismuth and "arsenic" injections are then resumed, the latter consisting of five doses each of 0.75 gram. In thirteen weeks, therefore, the patient has been given 6.3 grams of "914," 2.88 grams of bismuth metal (the dosage of bismuth being always reduced to the common denominator of bismuth metal), and about 650 grains of potassium iodide. The whole course is then repeated after the patient has had six weeks' rest. It is then repeated for a third time, with an interval of two months between the second and third courses. From the first to the last injection the patient is therefore under treatment for fifty-three weeks. Thereafter repeated clinical and pathological examinations are carried out for at least another year, in order to make certain that no signs of relapse occur after the cessation of active treatment.

The system recommended by Lees may be divided into eight three-monthly sections. During the first eight weeks 4.2 grams of "914" and 4.2 grams of bismuth (or 7 grains of mercury) are injected in weekly fractional doses. Then for two weeks 15 grains of potassium iodide are given t.d.s. by mouth. A fortnight's rest then precedes the commencement of the second period, which consists of 2.4 grams of "914" and 4 grams of bismuth (or 7 grains of mercury), and the fourteen days' potassium iodide. The third course contains no "arsenic," but consists of 3.6 grams of bismuth (or 8 grains of mercury), and the potassium iodide. The fourth section comprises 2.25 grams of "914" and 3.6 grams of bismuth (or 8 grains of mercury), and the iodide. The fifth period contains neither "arsenic" nor iodide, but only 3.6 grams of bismuth (or 8 grains of mercury). The sixth and seventh courses are similar in that they consist of 3 grams of bismuth (or 8 grains of mercury) only. The eighth and final period contains 3 grams of bismuth (or 8 grains of mercury), and the iodide. Therefore during two years' active treatment, 10.65 grams of "914" (this total includes occasional provocative doses) are injected, together with 28 grams

of bismuth (or 62 grains of mercury), whilst about $6\frac{1}{2}$ oz. of potassium iodide are administered by mouth. Subsequent observation over as long a period as possible is again advised.

In Burke's system the active treatment is condensed into thirty-four weeks, during which time he gives injections each week of "914" for six weeks, then six weeks' bismuth, then the "arsenic" again, and so on. He does not give "arsenic" and bismuth at the same time. Nevertheless, he contrives to administer 13.2 grams of "arsenic" in the form of stabilarsan, and 4.8 grams of metallic bismuth. Here again, however, subsequent observation of the most stringent clinical and pathological types is continued over a further period of at least two years.

In contrast with these vigorous courses is that devised by Whitfield, and somewhat modified by Drake and Thomson. They give one-grain pills of hydrarg. cum cret. t.d.s. by mouth (or $\frac{1}{8}$ grain of the yellow iodide of mercury) for two years without any intermission. "Arsenic" is given only once in every three weeks, and then for six injections only at the beginning of the course. But each dose consists of 0.9 gram of "914," *i.e.*, 5.4 grams within the first fifteen weeks of treatment. This total amount of "arsenic" corresponds approximately with that given during the same period in the other systems, but here it is never repeated unless there are signs of clinical or Wassermann relapse. Bismuth is not used as a routine, but only for cases which have proved themselves in any way resistant. They claim that prolonged observation of their cases gives a "cure rate" at least as good as those claimed for the more vigorous courses.

Before proceeding to a consideration of the points raised by this wide divergence, let us first glance at the total dosages given in a florid secondary case. In such circumstances Harrison gives at least four of the courses above described (instead of three), taking the Wassermann, etc., as guides, and administering three courses beyond the first, which has resulted in negative serum reactions. Lees' system also corresponds in that the same series of injections are given, but the total amounts of the drugs administered are now 14.25 grams of "914," 30 grams of bismuth (or 62 grains of mercury), and $10\frac{1}{2}$ ounces of potassium iodide. Drake and Thomson start with nine injections of "arsenic," *i.e.*, 8.1 grams of "914" in twenty-four weeks, the mercury being administered as before. Should the serum still give a positive reaction,

three more injections are given, and the blood again tested. Should the Wassermann still be positive, bismuth may be substituted, or the "arsenic" continued, the decision resting partly on the general history, etc., of the case and partly on the relative quantitative strengths of the Wassermans that have been performed. In brief, then, all the systems here involve heavier dosage, and in all it is the aim to get rid of signs and symptoms, and to produce a negative serum reaction as soon as possible. This negative reaction must then be maintained throughout subsequent treatment and observation. Any relapse is the immediate sign for the reversion to more vigorous methods. Whilst the "arsenic" is usually given intravenously, it must be not forgotten that intramuscular injection of such sulphur preparations of "914" as sulphostab or sulfarsenol can be substituted. Their use may be rendered essential by the smallness of the veins, by the youth of the patient, or when it is desired that the "arsenic" shall be less rapidly excreted from the body.

In contrasting these two (high and low) dosage systems, assessment is very difficult. There is no doubt that an otherwise healthy young adult suffering from early syphilis can tolerate 0.9 gram of "914" with perfect safety, although this fact is still disputed in some quarters. There is equally no doubt that an interval of three weeks between injections does render the arsenical treatment safer in that that interval allows more time for the more complete excretion of "arsenic," so that any danger of its accumulation is avoided. On the other hand, it is true that by far the greater part of the "arsenic" is excreted within three days of intravenous injection, and that the liver may be protected to some extent by feeding before injection, instead of starving. In spite of this fact, it would seem that the high dosage method does involve greater risk to the patient. The risk might, however, be considered justifiable if the reported results were definitely better than those obtained by Whitfield's method. But this does not seem to be true, unless further experience leads Drake and Thomson to reconsider their position. This is unlikely to occur, as they have recently reviewed all their old cases with whom they could get into touch, a review which apparently confirms their previous attitude. Unfortunately very few practitioners rely on the low total dosage, so that the total numbers of cases treated by the two methods are not strictly comparable. This difference between the two systems

might be summarised by the question: "Are the spirochætes more likely to be killed by relatively infrequent high doses of '914' or by more frequent lower doses?"

Looking at the use of bismuth and mercury, we again have extremes. On the one hand, bismuth alone is used as a routine; on the other, mercury alone. Between the two all gradations of opinion are represented. It is true that the intramuscular injection of either metal does ensure its presence in the tissues of the patient, whereas oral administration must in the first place be dependent on the co-operation of the patient. Localisation of the injected material is so rare that it can have no weight as an argument against injection. On the other hand, the benefits derived from mercury may be dependent on the presence of a mercurial salt, an objection which is used against hydrarg. cum cret., but which does not hold where the iodide is given. To doubt that such administration produces any definite effects is merely to doubt the experience of all physicians who treated syphilis in the past. To go so far as to say that mercury is completely valueless, as has been recently stated, is merely to maintain that no case of syphilis was ever cured before the introduction of bismuth, a manifest absurdity. The real objection to the oral medication is that it must depend for its effects on the goodwill of the patient. It is just as easy to obtain this co-operation as it is to get a patient to attend weekly in order to have an injection, for very few injections are entirely painless.

This brings us to one of the most important factors in the treatment of syphilis: it is in fact probably the most important. In the management of all diseases it is essential to gain the confidence of the patient, but in no disease is that confidence more essential than in early syphilis. The administration of "arsenic" removes all the signs and symptoms so very quickly that the patient feels perfectly well. It must be difficult for a layman in such circumstances to believe that months of treatment are thereafter necessary. Also nowadays one only too frequently meets a suspicion that the hospital or the physician is prolonging matters, either for financial or "experimental" gain. Continued attendance at the clinic must therefore be gained by means of a simple explanation of all facts and a sympathetic discussion of difficulties raised by the patient. When reasonable methods fail, stronger psychological persuasion must occasionally be used. "Must be used," because the disease

is communicable both directly and to the next generation. For these reasons, quite as much as because of the manifold aspects of syphilis, it is important to realise that every case must be considered and treated as an individual. It is sometimes difficult to remember this point when dealing with large numbers in a clinic, but a falling-off in the attendances is sure to refocus attention. By then, however, much irreparable damage will have been done.

It would seem that the establishment of clinics all over the country, and the widespread use of intensive treatment, must have reacted considerably on the incidence of syphilis. It is true that the number of early cases has dropped markedly since the years immediately succeeding the War, but the disease is still common, and there has been no very definite decrease during the last two or three years. The incidence rate now seems to be more or less stationary. But even this state of affairs has only been achieved with difficulty, for the last few years have seen a marked increase in the amount of treatment given to any individual case, not only in this country, but also on the Continent. That is, to produce the same clinical and pathological results as were obtained in 1920, most workers have found it necessary to double or even treble the amount of arsenical dosage. For example, twelve years ago a sero-positive primary case would almost invariably give a negative serum reaction after three doses of 0.9 gram of "914," and this negative reaction did not vary throughout subsequent observation. To-day at least six such injections, and often more than six, are needed to attain a negative Wassermann in very many cases. Even then a serological relapse frequently occurs, so that even more active treatment has to be given. It is necessary to stress that this experience is getting increasingly common in all countries. But an even more alarming phenomenon is becoming apparent. There have always been a few resistant patients, but during the last two years not only has the number of serum-resistant cases increased, but we are seeing more and more examples of severe clinical relapse. The Continental literature contains many striking illustrations, and in this country Semon, Drake and Thomson have reported some seventeen cases between them during the last nine months. To quote one instance in detail, an instance which can be added to the above number. Last July, an otherwise healthy man of twenty-four years of age presented himself at hospital with a primary chancre from which spirochaetes were

obtained. His Wassermann was negative, and there were no clinical signs of generalisation of the disease. He was given mercury by mouth regularly, and four injections of 0.9 gram of "914" at three-weekly intervals. Two days after the last injection, that is within ten weeks of his first attendance, he showed definite gummatous ulceration of the right arm and left ankle. It is now four months since these appeared, and whilst they are healing, progress is remarkably slow despite injections of various arsenical and bismuth preparations, the administration of iodides, etc., and careful local dressings.

It is admitted that this is an exceptionally severe example, yet such cases are occurring in widely separated areas, and in clinics where every variety of treatment, both as regards dosage and preparations, is in use. Thus, although modern methods have undoubtedly proved a great advance on the older forms of treatment, they do not yet provide us with a certain means of rapid cure. Prolonged treatment, close observation and all the therapeutic skill of the physician are still essential. They have not been replaced by any guaranteed or "fool-proof" system.

CHAPTER VIII.

JAUNDICE IN INFANCY AND CHILDHOOD

BY

REGINALD LIGHTWOOD, M.D., M.R.C.P., D.P.H.

Assistant Physician for Diseases of Children to the Westminster Hospital; late Medical Registrar and Pathologist to the Hospital for Sick Children, Great Ormond Street.

WHAT differences, it may be asked, are there between jaundice in the adult and jaundice in the child that call for discussion. Obstructive jaundice, common enough in later life, is rare at all periods of childhood because of the virtual absence of carcinoma and the infrequency of gall-bladder disease. In the neonatal period the causes of jaundice differ so widely from those of childhood that the subject falls naturally into two parts.

The Neonatal Jaundices.

Physiological Jaundice, or simple icterus of the newborn, occurs in some 33 per cent. of all infants. It is accompanied by only one symptom, and that is drowsiness. It requires no treatment, and is chiefly of interest to the physiologist. In explanation of physiological jaundice, it is conjectured that the following changes occur at the beginning of extra-uterine life. The oxygenation of the unborn infant depends, through the placental circulation, on the mother. The placenta as a respiratory organ is less efficient than the lung, and foetal respiration is also hindered by partial mixing of arterial and venous blood in the foetal heart and circulation. To compensate for this state of affairs the foetal blood is supercharged with red cells and hæmoglobin. At the moment of birth 5.5 to 6.0 million red cells and some 120 and 140 per cent. of hæmoglobin may be regarded as normal. After birth this amount of hæmoglobin is in excess of the infant's needs, and a brisk hæmolytic process comes into play, the mechanism of which is not under-

stood. The degree of jaundice which appears at this time may be correlated with the loss of hæmoglobin and cells occurring during the first ten days, and a positive indirect van den Bergh reaction, indicative of hæmolysis, is found. That this explanation is incomplete is proved by the fact that examination of foetal blood from the umbilical cord at the time of birth shows already a high content of bilirubin, and it is probable that the hæmolytic process begins *in utero*. It is important to note that this physiological jaundice accompanies a fall from the high foetal hæmoglobin concentration to a figure which is within normal limits for early infancy and anæmia does not result.

In *premature infants excessive hæmolysis* may occur with prolonged icterus and resulting anæmia. Occasionally a transfusion of blood will be of benefit in a severe case. After the excessive blood destruction is at an end, such premature infants are apt to run short of iron, and a course of saccharated sugar of iron (grains 2 to 5) becomes indicated.

A still more violent hæmolysis occurs in *icterus gravis neonatorum*, a disease which may or may not affect several other infants of the family, and has a high mortality. The clinical features of icterus gravis are important because it is easily confused with congenital obliteration of the bile ducts, congenital syphilis, and with other diseases. Icterus gravis is present at birth, or becomes apparent very soon afterwards. Unlike simple icterus of the newborn it results in a sick child, and a more or less severe hæmolytic anæmia quickly develops. The liver and spleen become enlarged, and a characteristic blood picture may be expected. Large numbers of nucleated red cells and reticulocytes are seen, together with a few myelocytes. As in all grave jaundices, there is a tendency to bleeding, and hæmorrhages into the skin, from the umbilicus or over the surface of the brain, may occur. Nervous manifestations, such as drowsiness, convulsions and spasticity, arise in some of the cases. For the treatment of this disease intramuscular injections of human serum have been recommended, but I have found transfusions of citrated whole blood more valuable.

Congenital obliteration of the bile ducts is a uniformly fatal disease, and our consideration need go no further than its diagnosis. The jaundice appears from the first to the third week of life, and steadily increases, though an ordinary physiological icterus developing about the third day may pass imperceptibly into the obstructive

jaundice of biliary atresia. The stools are pale from birth, though exceptionally they may be coloured by bile excreted directly into the large intestine. In such a case a pale stool covered with a shell of coloured material is passed. The urine always contains bile, and the van den Bergh test gives a positive direct reaction. A fatal issue may be expected between the fifth and seventh month of life, but occasionally it is postponed as late as a year.

Congenital Syphilis is a very occasional cause of jaundice in the newborn infant, so that it is wise to exclude this possibility if there is anything to suggest that the diagnosis is other than physiological icterus. A negative Wassermann reaction is not infallible during the first few weeks of life, and more reliance is to be placed on examination of the blood of both parents. An X-ray of the long bones of a syphilitic infant is often a valuable method of establishing the diagnosis. Severe neonatal jaundice is a grave sign in congenital syphilis.

Infection of the umbilicus may lead to jaundice, but only in severe cases. The temperature is elevated and the liver is enlarged, but not the spleen, unless systemic infection has occurred. Treatment should be directed to the umbilicus, and a guarded prognosis given, though recovery takes place in many such cases. The commonest causes of neonatal jaundice may be summarised as follows :

Type	Onset of Icterus	Degree	Duration
Simple physiological icterus	About third day	Slight	7-14 days or or less
Neonatal hæmolysis of prematurity	About third day	+	About three weeks
Neonatal sepsis with jaundice	After first week	++	Variable
Congenital syphilis	Variable	++	Variable
Icterus gravis neonatorum	At birth or soon after	++	Up to eight weeks
Congenital obliteration of bile ducts	First to third week	+++ Progressive	Until death

Jaundice in Later Infancy and Childhood.

Catarrhal Jaundice is the commonest variety of jaundice after the first year. There are some who hold that this type may occur also in the neonatal period. If we restrict the term catarrhal jaundice for the mild hepatitis which is sometimes epidemic and

commonly supposed to result from an unknown virus infection, then the conception of its occurrence in the neonatal period is improbable. Moreover, it is contrary to what we should expect of the comparative immunity of the newborn infant to many virus infections and to the rarity of this type of jaundice in later infancy. In children past the age of one year catarrhal jaundice is increasingly common. Its treatment requires rest in bed and a light diet relatively free of fat and protein and rich in carbohydrates, particularly sugar. The bowels should be gently opened with cascara, senna or rhubarb, and a small dose of sodium salicylate or hexamine may act beneficially in the biliary channels. The following prescription is suitable for a child of five :

{	R	Sodii salicyl.	gr. v
		Sodii bicarb.	gr. v
		Inf. rhei.	m. xv
		Syrup zingiberis	m. x
		Aq. menth. pip. ad	℥ ii t.d.s. p.c.

Though the prognosis in catarrhal jaundice is almost uniformly good, two very serious sequels may occur, and cannot be foretold. First, the attack of jaundice may be repeated at intervals until a state of *hepatic cirrhosis* is set up. We know of nothing to prevent this misfortune, though drainage of the gall-bladder is perhaps worth a trial. Secondly, an attack of catarrhal jaundice, clinically indistinguishable from any other, may take a turn for the worse just at a time when one expects improvement to occur. At the first warning of this the wise practitioner will do well to seek a consultation, for such cases may pass into a state of grave jaundice, with vomiting and cerebral symptoms, and die of cholæmia within ten days of the onset of an apparently trivial illness. The underlying pathology of such as these is that of an acute hepatic necrosis or yellow atrophy (red atrophy if hæmorrhagic). It is quite undecided if these fatal cases are severe forms of simple catarrhal jaundice or an entirely different disease, but of their initial similarity there is no doubt.

All other forms of jaundice are rare in childhood. Idiopathic hepatic cirrhosis and some cases of splenic anæmia may be made manifest by an attack of jaundice. A simple pneumonia, as well as other specific infections, may be complicated by icterus without the prognosis being necessarily the worse. Before leaving the infective group of jaundices I will allude to the significance

of jaundice appearing a few days after an attack of appendicitis. Such an occurrence may herald suppurative pylephlebitis, which is nearly always fatal.

Among the hæmolytic jaundices of childhood we need only concern ourselves with acholuric family jaundice. Although cases have been recognised within a few days of birth, this disease does not usually show itself until the period of the second dentition. The main clinical features are those of a chronic hæmolytic anæmia. The skin is pale and yellow, not unlike that of pernicious anæmia. The spleen is enlarged, but not the liver or lymphatic glands. The urine contains urobilin and is free of bile. Examination of the blood shows an anæmia in which the colour index is usually as high as 0.8. Many reticulocytes will be found in vitally stained smears (cresyl blue), and the presence of a few normoblasts is rather suggestive. The white cells and platelets are normal. The diagnosis may be confirmed by testing the resistance of the red corpuscles to hypotonic saline. Some families of acholuric jaundice do not give this test. Probably there is no other medical condition in which splenectomy is so strongly indicated. Any affected child of five years and upwards may be expected successfully to withstand the operation. In practice, severe anæmia is the chief contraindication, though it should be only a temporary one, and transfusion of blood before operation will be required if it is much in evidence. The operation should not be long delayed, because its optimum moment is in the pre-anæmic stage. A knowledge of the degree of blood destruction and compensating marrow activity may be gained by keeping a watch on the red cell count and the percentage of reticulocytes. In this way a timely intervention may anticipate dangerous anæmia.

CHAPTER IX.

THE

ÆTIOLOGY AND TREATMENT OF

DISSEMINATED SCLEROSIS

BY

R. S. ALLISON, M.D., M.R.C.P.

Assistant Physician, Royal Victoria Hospital, Belfast ; Consulting Physician,
Benn Ulster Eye, Ear and Throat Hospital.

DISSEMINATED sclerosis is one of the common organic nervous diseases. In England and Wales, 788 deaths were attributed to this cause in 1930. In a large district at least one case may be found for every five to ten thousand of the inhabitants. There were 106 cases among 728 organic nervous diseases admitted to the Royal Victoria Hospital, Belfast, in three consecutive years.¹ The sexes are affected about equally, and the onset is usually between the ages of twenty to thirty-five.

The onset of symptoms is often related by patients to injury, influenza, or some other infectious disease, and in women to pregnancy and parturition. Women afflicted with the disease often feel better during pregnancy and worse afterwards, and cases have been observed where the first symptoms arose after delivery. It is true also that when a limb has been injured previously, its function is likely to be affected first, before other parts are involved, but this is not invariable. A certain number of cases appear to arise genuinely out of an obscure illness to which the term "influenza" is often applied. Examples of this kind are, however, in the minority. In the greater proportion, a careful history will show that there have been earlier and forgotten symptoms. It is obvious, therefore, that the effect of these alleged causes is secondary rather than primary, and that they act largely in unmasking a hitherto latent disease.

Interest has been aroused in recent years over the geographical distribution of the disease. It appears to be more prevalent in

some countries than in others. Switzerland and Ireland have a high incidence, but the exact study of this problem is hindered by the fact that the disease is not notifiable, and is not classified separately in the international list of causes of death. Now that its frequency is better recognised it is to be hoped that these obstacles may be removed. A point of interest in connection with this aspect is the resemblance which exists between the geographical distribution of disseminated sclerosis and goitre. This has been observed in the United States,² and the frequency of the nervous disease in Switzerland has already been referred to. It is not uncommon, too, in my experience to find disseminated sclerosis and goitre in members of the same family.

At first disseminated sclerosis was thought to be a primary sclerosis. The usual findings at autopsy were patches of sclerosis or hardening scattered throughout the brain and spinal cord, and chiefly affecting the white matter. It is now recognised that this sclerosis is but the end result of a morbid process, and not a primary lesion. It is a localised overgrowth of connective tissue or neuroglia to replace a damaged structure.

The essential feature of the pathology is a strictly localised and scattered perivascular demyelination. The splitting up of the myelin sheaths which normally surround nerve fibres is accompanied by a local inflammatory reaction. It is not yet decided whether the demyelination is the result of the inflammatory reaction or the cause of it. Two views appear to be likely, and to challenge solution.

(1) *The process is primarily an infective one, the organism having a selective action on myelin.*—If this view be correct it is probable that the route of infection is by the blood, and that the agent responsible may be present in the cerebro-spinal fluid and perivascular spaces. So far the search for a causal organism has been inconclusive. Some years ago a claim was made for a spirochæte which is described as being thicker and coarser than the spirochæta pallida. In recent years a group of acute infective diseases of the nervous system has attracted attention in which demyelination is a characteristic histological feature. Post-vaccinal encephalitis, post-measles encephalitis and acute disseminated encephalo-myelitis are examples of this kind. The possible relationship between disseminated sclerosis and this group has been under consideration. In the acute vaccinal type, convulsions,

paralysis and fever supervene about twelve days after vaccination. Histologically there is a perivascular cellular exudate and demyelination. The lesions are indeed similar in many ways to those seen in early disseminated sclerosis. A proportion die, but in those who recover a remarkable feature is the degree of recovery which takes place, and there is immunity to further relapses. The inference has been drawn, therefore, that "variations in dosage and virulence of the infective agent and in the immunity of the patient may account for the wide range of reactions which we regard clinically as different diseases."³ This aspect of the infective theory is a fascinating one, and shows great promise.

(2) *The morbid process is primarily endogenous.*—That other factors as well as infection can cause demyelination has been shown by the recent work of Mellanby, who produced it in the spinal cords of experimental animals fed on diets deficient in vitamin A. Such a gross deficiency in diet could scarcely occur in man under ordinary circumstances. Although this finding, therefore, is not directly applicable, it opens up the possibility of the demyelination being due to a deficiency rather than to a bacteriological cause. In addition to an extrinsic factor there may be an intrinsic defect or instability of the myelin which makes it unduly vulnerable and liable to split up under certain circumstances. From this point of view the local inflammatory reaction would be secondary to the demyelination: the response to a local chemical irritant in the form of disintegrated myelin. This view is supported by the remissions and relapses so characteristic of the disease. As is well known, they often coincide with intercurrent infections, injury and parturition. In this sense, as in the infective, these events may be regarded as activators of a dormant condition. If there is an endogenous factor in a disease one expects to find a familial tendency. Although it is rare to find more than one member of a family attacked, a neuropathic inheritance is fairly common. Gowers held this view, which is supported by recent work. There is some evidence also to suggest that the subject of disseminated sclerosis presents a definite physical and mental type, and that from early youth he may have been different from his fellows. This is well illustrated in the "Journal of a Disappointed Man," by Barbellion, which gives a good and intimate account of the life history and personality of a subject of the disease. In helping to understand the clinical course of the disease it is useful

to remember that the important structures of the nervous system, the nerve cells and their processes, are not directly affected. Thus, in the early stages, even in the presence of a severe degree of loss of function, partial or even complete recovery of function is always possible, and, indeed, may be confidently expected. The functions of the nerve fibres passing through the area of demyelination are temporarily interrupted, but as the inflammatory reaction subsides complete restoration of function may result. More often, however, the denuding of the fibres of myelin or the formation of dense neuroglial tissue (sclerosis) leads to some degree of permanent impairment. After the first onslaught has had time to subside, a few weeks, months, two, five, or even twenty years may elapse before the process is repeated, often in a different part. This continues, the degree of recovery becoming less and less until a chronic stage is reached with permanent paraplegia. The factors which govern the march of events in different cases are unknown, but clearly they vary considerably, no two cases being the same. This clinical feature of variability encourages us in the belief that in treatment we are not necessarily dealing with a hopeless problem whose progress we are powerless to arrest. The general trend of the disease is downhill, but cases do occur in which the symptoms are arrested for years, and permanent remission is not unknown. The illustration is an example of a typical case :

B. T., female, aged forty-three.

Ages 18.—Temporary blindness left eye.

30.—Left foot dragging ; paræsthesia ; recovery.

34.—“ Nervous breakdown ” ; recovery.

35.—Lame in left leg ; symptom persisted.

36-43.—Gradual increasing weakness of lower limbs ; spastic paraplegia ; intention tremor present in upper limbs ; pallor of optic discs.

Treatment.

It is obvious after considering the above that we must be on our guard against attaching too much importance to the action of a drug in relieving symptoms. Any remedy may seem to do good in the early stages by coinciding with the speedy recovery of the patient or natural remission of the disease. Thus a young man may lose the power of a leg and regain it within the space

of three months, no matter what treatment he is following. Even in the later stages, where apparently permanent paralysis has supervened, a temporary improvement is not unusual. The only reliable test of therapeutic efficiency is that of time. We shall be on surer ground if it can be shown that a given drug has led to uniform arrest of symptoms in a large group of cases for a period of five to ten years. None of the methods of treatment now given are specific in this sense. They represent fairly, however, the lines on which present-day treatment is conducted, and their relation to the ætiological principles will readily be seen. There are three chief lines of treatment :

(1) *The introduction into the circulation or tissues of agents calculated to produce a febrile reaction and to stimulate the general defensive mechanism against infection.*—This method has been used in many chronic diseases with some measure of success. To this class belong treatment with intravenous injections of anti-typhoid vaccine (T.A.B.), intramuscular injections of sulphur preparations, and inoculation with malaria. The first two methods are described.

Anti-typhoid vaccine : A course of three to six intravenous injections is given at five-day or weekly intervals. The initial dose is cautiously chosen, 15–30 million, and diluted in 2 c.c. of normal saline. A sharp reaction may follow, with rigor, and the temperature rises rapidly (100°–105° F.). If the injection is given in the morning the temperature will have reached its maximum before nightfall and have subsided by the following morning. Occasionally it is prolonged to forty-eight hours, or there may be little or no reaction. The next dose is altered accordingly, and gradually increased to a maximum of 150–250 million.

Sulphur preparations : “Sulphosin Leo” or “Colsul” may be used. Both are made up in ampoules and consist of 1 per cent. sulphur in aqueous or oily solution, to be injected intramuscularly. Fever is produced, and reaches its maximum within twenty-four hours, rapidly returning to normal. The injections should be given in the evening, and the temperature recorded hourly the next day from morning till evening. The initial dose is 1–2 c.c. If this is ineffectual, 3 c.c. may be given in four days’ time, when a good response is almost certain. For the third injection the same dose or 5 c.c. may be given. Four to six injections at intervals of four or five days constitutes a course. The site of injection may be the buttock or outer side of the thigh. A different place is chosen each time.

The point of the needle should be inserted until it touches bone, and then withdrawn for a quarter of an inch or so, and the injection made. The sites of injection are often painful afterwards, but no serious local reactions have been seen when the dose is not greater than 5 c.c.

(2) *Drugs given for their bactericidal or specific effect.*—Pierre Marie advised *mercury* for the infective element in the disease, and *potassium iodide* for the sclerotic element. They are not much used nowadays. The use of *quinine* has recently been revived. This was formerly advocated by Williamson, of Manchester. *Hexamine* may be prescribed in early cases with a sudden onset in doses of ten grains thrice daily for a fortnight. *Arsenic* has probably been used more extensively than any other drug in the treatment of disseminated sclerosis. Fowler's solution was formerly the favourite preparation. To-day this method has been superseded by the intravenous use of novarsenobillon (914), and silversalvarsan. These drugs were introduced in response to the spirochætal theory, though so long ago as 1911 Farquhar Buzzard advised their use. Silversalvarsan possesses no clear pharmacological advantage over the simpler novarsenobillon, and is more difficult to administer owing to its dark-black colour in solution. A course of novarsenobillon would consist of six to eight weekly injections of 0.15, 0.15, 0.30, 0.30, 0.45, 0.45, 0.45 gm. Larger doses are not indicated, and may prove harmful. If silversalvarsan is used a suitable course would be 0.05, 0.05, 0.05, 0.10, 0.10, 0.10 grm. When giving arsenic intravenously the usual precautions are necessary as regards technique, and toxic symptoms should be watched for. Their appearance, even in a mild form, calls for smaller doses or abandonment of this method of treatment.

(3) *The administration of preparations of vitamin A.*—This is of recent origin, and has a rational basis. Various preparations are available, and need not be enumerated here. The raw juice and pulp of liver obtained by sieving is as good as any. Cod-liver oil, green vegetables, lettuce and bananas are also rich sources of vitamin A, and should be included in the diet.

Rest, massage, electricity and general management.—Compulsory rest is inevitable in the later stages, but it is often asked how much rest and exercise should a patient be allowed in the ambulatory stage. Undue fatiguability of gait is often an early symptom of disseminated sclerosis, and during remissions, when the power

of the limbs is reasonably good, the patient can walk some distance, but then the leg begins to drag. Patients should be advised, therefore, in the early stages to limit their exercise and to avoid over-fatigue. A short rest in bed with daily exercises will often do good in improving the gait. Massage is also helpful in maintaining muscular nutrition in bed patients and in alleviating spasticity. *Electrical treatment* by Faradism or galvanism is rarely indicated, and patients do not like it. Passive and resisted exercises, with the repetition of simple movements designed to promote muscular co-ordination, are of value in the partly disabled and in those showing signs of recovery from a recent relapse. The general health of patients suffering from disseminated sclerosis is usually good. The presence of obvious foci of infection in the teeth and nasal sinuses, throat and urinary tract will naturally receive attention, though too energetic measures in this direction are to be avoided.

REFERENCES.

- ¹ Coates Foster : *Brit. Med. Journ.*, 1,539, 1933.
- ² Davenport, C. B. : Asscn. for Research, N.Y., Report on Multiple Sclerosis, 2, 43, 1921.
- ³ Brain, R. W. : *Quart. Journ. Med.*, 343, 1930.

CHAPTER X.

BILIARY COLIC

BY

CHARLES NEWMAN, M.A., M.D., F.R.C.P.

Assistant Physician, King's College Hospital; Assistant Registrar, Royal College of Physicians.

BILIARY colic shares with many other medical emergencies the need for foreknowledge: a doctor must know exactly how to recognise and treat it, even though, as is quite likely, he has never seen such a case in his student days. It is difficult otherwise to avoid the necessity of learning at the expense of the patient's suffering. The emergency, from the point of view of the patient and the relatives, is one of the most extreme in medicine; it is doubtful if a pain could be more excruciating without causing loss of consciousness. Biliary colic is so uniquely terrible that the term is, as a rule, restricted to the pain caused by a gall-stone in the bile ducts. And quite rightly so, because although the pain of cholecystitis and of biliary dyskinesia may be severe, it never quite amounts to that of gall-stone colic. That the biliary pains should resemble one another is only natural—they are all due, like the pains in other hollow viscera, to distention. The pains produced by different causes vary in intensity rather than kind, in accordance with the degree and suddenness of this distention. Nothing distends the extra-hepatic biliary system so much in so short a time as the complete obstruction by an impacted stone. The part played by muscular spasm is doubtful; it is certainly less important, as the immediately painful factor, than distention (because the colic of a stone in the neck of the gall-bladder is neither so common nor so severe as that due to a stone in the ampulla of Vater, and colic may arise from impaction in the non-muscular common bile duct). But the gripping of the stone by the muscle of the ampulla is the cause of the completeness of the obstruction, and the relaxation of this spasm ends the extremity of the pain.

The Gall-Stone.

As gall-stones are commoner in women, so is biliary colic more often seen in women than in men. The type of stone responsible is the facettèd stone produced by a previous attack of cholecystitis. Pure cholesterol solitaires rarely cause colic: they are too large. Calcium bilirubin stones are an unusual cause, partly because they are themselves rare, and partly because they are so heavy that they sink to the bottom of the gall-bladder. They are, however, the usual cause of biliary colic in patients with acholuric jaundice. Recurrence of gall-stone colic, after cholecystectomy, if it is genuine (it is nearly always dyskinetic in origin), is due to "earthy" stones which form in the ducts, and not only in the gall-bladder, as do the other varieties of stone. Facettèd stones become more common with advancing age, but colic does not. That is to say, if a person with gall-stones passes the age of sixty, increasing hypokinesia of the gall-bladder progressively lessens the risk of stone migration. Biliary colic is the complication of the younger person with the more active biliary apparatus who has had the misfortune to develop gall-stones.

The Attack.

Biliary colic tends to come on in the night rather than by day, sometimes with a definite association, such as pregnancy (possibly because of increased vagal irritability), or after a dietetic indiscretion (especially with respect to fats, which stimulate biliary motility), but very often for no known reason. The pain starts in the mid-epigastrium, the bile ducts and liver being embryologically of mid-line origin, but it so soon becomes increasingly severe over the gall-bladder that its original site is either forgotten or will at any rate be unlikely to confuse the diagnosis. The true colic is situated between the right costal margin in the umbilicus, nearer to the ribs than the gall-bladder point, which is mid-way between the two. It is a typical colic in that it remits and intensifies, the paroxysms lasting for minutes on end. The pain radiates characteristically to back and *upwards*, but also very often *across to the left*, as in simple dyskinesia. The commonest point of radiation is to the angle of the right scapula; the "tip of the shoulder" pain is due to diaphragmatic irritation, and is more characteristic of amoebic abscess than of gall-bladder disease. It is actually more common in cholecystitis than in gall-stones, and has been

over-stressed in the past because it is so remarkable. During the paroxysms the patient rolls about in ^{severe pain} agony, unable to obtain relief from position or pressure, and oblivious ^{of the} of the cold, or even of falling out of bed. Breathing is interfered with by the emotions: the patient may hold her breath until she begins to become cyanosed. Screaming and grinding the teeth during paroxysms give place to gasps and groans during the remissions. The patient usually sweats profusely. The pulse is rapid, and becomes weak; some degree of collapse is the rule, and the patient may pass into a state of shock or actually faint. Interspersed with the paroxysms there are often rigors with chattering of the teeth. Air-swallowing and flatulence are very common in a vain attempt to find relief. The intensity of the pain often leads to vomiting, and after a vomit the attack sometimes subsides, either finally or temporarily. The temperature is almost always raised above 99° F., and is often high, without necessarily any evidence of infection; it is probably of hepatic origin, like the pyrexia of liver necrosis and of secondary carcinoma of the liver.

Examination.

Examination is very difficult, and a routine examination for purposes of exclusion is impossible, but between the paroxysms one can usually find the positive signs. It will be found that the liver region, including the ribs overlying the liver, is tender, and that the upper right rectus is rigid. The mid-point between the ninth costal cartilage and the umbilicus is especially tender (pressure increases rather than relieves the pain). The liver is often swollen, tense and rather blunt-edged, but in a fat woman such minutiae cannot be expected. Visible jaundice, on first seeing the patient, is unusual, partly because it takes at least hours to develop and partly because jaundice is difficult to see by artificial light; but next morning there is often a perceptible yellowness of the conjunctivæ. An increase of bilirubin in the blood is to be found in all cases except those due to a stone in the cystic duct (which is rare), but this may not lead to visible jaundice, or to the appearance of bile in the urine. The van den Bergh test is invaluable for this reason, and all the information required may be very simply obtained. The increase of bilirubin consists of the prompt reacting type of pigment, which is never found in normal serum. All that is necessary is to obtain a little blood from a vein with a syringe

and hypodermic needle, allow it to clot, and float a little diazo-reagent on to the surface of the serum in a small test tube. A pink ring at the junction of the two is, in such a case, almost conclusive proof that it is biliary colic. The two solutions from which the reagent is made keep indefinitely (although the reverse is often stated), and the test is quite easily done by any general practitioner, especially if he has taken the opportunity of having learnt to do it during his student days. The method is, like finding the gall-stone in the fæces, a subsequent proof of the diagnosis, not a preliminary to treatment.

The attack lasts for hours if untreated, and fades away gradually; sometimes the end comes abruptly, but in any case the patient is left worn out, exhausted, and with a sore feeling over the right hypochondrium which lasts as long as the attack of colic, or longer. The patient usually sleeps profoundly after the attack is over, but there is always a risk of its sudden return at any moment.

Differential Diagnosis.

This is in practice much easier than it might seem on paper, but there are one or two important points.

(1) *From the other colics* the diagnosis is easy. If biliary colic ever radiates downwards (and anything may happen in medicine), such an event is exceedingly rare, and no other colic radiates upwards. This radiation of the pain is the principal differential feature. The associated alimentary symptoms of gastric or colonic disease, together with the main point of tenderness and pain, will alone serve to indicate the organ in which the colic is arising, and the positive signs of biliary colic usually make mistakes avoidable.

(2) *From Duodenal Ulcer*.—The night pain, its situation, and the tenderness in the right epigastrium, might seem very like the picture of duodenal ulcer, but in practice the difficulty is in distinguishing between *chronic* gall-bladder disease and an ulcer. The distinguishing point in an acute case is that the pain of an ulcer is repeated night after night and in relation to meals; biliary colic is only an occasional disaster.

(3) *From Acute Cholecystitis*.—This is very important, because the resemblance may be close and the treatments should be diametrically opposite. Cholecystitis may start very suddenly with pain like biliary colic. The pain is not usually so severe, but the pyrexia and rigors of sepsis naturally resemble those of

colic. The differences are that cholecystitis is, clinically, a disease of the gall-bladder itself; tenderness is greater than pain, the gall-bladder is swollen, but not the liver, and the pulse, though rapid, is full, not weakened. There is no jaundice with cholecystitis.

(4) *From Myocardial Infarction*.—The syndrome of myocardial infarction is characteristic, provided the pain is in the left chest and arm; but sometimes the pain is epigastric in site. Then the associated collapse, vomiting and sweating may simulate biliary colic. The distinctions are these: The pain is left-sided rather than right, and is continuous; there are no signs over the gall-bladder region; the temperature is always subnormal in the early stages, whereas in biliary colic it is raised, and there is some degree, often severe, of dyspnoea. Signs are found in the heart rather than the abdomen, a high diastolic pressure (of hyperpiesis) being especially characteristic. The patient is older (over fifty) and usually a man.

(5) *From Pneumonia*.—A well-known difficulty, but a common cause of error. The association of the pain with respiration and the raised pulse-respiration ratio are more useful than the signs at the base of the lung, which are often very slight in the early stages.

(6) *From Acute Pancreatitis*.—This may be difficult, because acute hæmorrhagic pancreatitis is in part due to a gall-stone in the ampulla. But the central pain, which is continuous, the extreme shock and the cyanosis of the finger-tips should lead one to suspect pancreatitis as an added complication.

Treatment.

The first aim, after making a diagnosis (or, indeed, before making a *certain* diagnosis, so long as one can be satisfied that the case is not one of abdominal suppuration), is to stop the pain, even before putting the patient to bed. The usual treatment is a hypodermic injection of $\frac{1}{2}$ gr. of morphine. Short of morphine or anæsthesia, no analgesic is powerful enough to stop biliary colic, but morphine has the grave disadvantage of being a vagal stimulant. Now the usual cause of biliary colic is spasm of the vagus—innervated muscle of the ampulla of Vater, and further stimulation of the vagus is precisely the thing to be avoided. The action of remedies directed to the cause is not rapid enough to make them advisable on humanitarian grounds, but it is wise to give atropine sulphate, gr. $\frac{1}{50}$, with the morphine to paralyse the vagal nerve endings as much as possible. Keep the patient under atropine to the extent of definite

dryness of the mouth for twelve hours. Apply tepid sponging if there is the least indication for it ; the temperature tends to rise when sweating is stopped by atropine, and sponging makes the patient much more comfortable.

As to alternative treatments : Hot fomentations to the liver, and a drachm of sodium bicarbonate in half a pint of warm water may relieve a mild case. The morphine derivative "Dilaudid" is said to have all the advantages of morphine without being a vagal stimulant, and is well worth a trial. It is now obtainable in this country. There is yet another treatment which I have not yet had the opportunity of trying, but which seems to me to offer great possibilities. This is intravenous anæsthesia with Evipan sodium. I do not put it first, because we do not know enough about its safety, especially with regard to its action on the liver, to make it a certainly safe recommendation, but I would suggest the following procedure : inject a solution of Evipan sodium (it is put up ready in ampoules) into the arm vein, slowly, until the patient goes to sleep, a matter of only a few seconds ; then inject another 2 c.c. This will give at least twenty minutes of anæsthesia, in which the patient can be put to bed, and further treatment applied. Occupy the relatives with the preparation of a large hot fomentation for the liver region, while you inject atropine sulphate, gr. 1/50, subcutaneously. This will paralyse the vagus, after which morphine can be given, if necessary, with far greater success, and may possibly be avoided. That can be determined when the patient comes round from the anæsthetic, by which time the atropine will be in action.

Finally, concerning the question of operation. Although it does not enter into the question of this emergency, strictly speaking, it will shortly concern the question of prevention of another emergency. If the stone is not passed, the patient is in danger of two things : a further attack of colic, or an ascending biliary cirrhosis, "the liver of surgical delay." Do not delay or oppose operative treatment unless you are certain that the patient is a *wholly* unjustifiable operative risk. The risk of not operating is very great, far greater than most physicians realise. No patient with a stone in the ampulla should get up after an attack of biliary colic with her gall-bladder untouched. There are few more distressing circumstances than to feel, after the patient has died of liver function failure or cholecystitis, that if only one had advocated an operation all might have been well.

CHAPTER XI.

BLOOD TRANSFUSION

BY

NORMAN HILL, M.D., M.R.C.P.,

Physician to the Metropolitan Hospital ; Senior Physician to the Belgrave
Hospital for Children, etc.

COMPARATIVELY few years ago a blood transfusion was regarded as a procedure only to be undertaken by a specialist in that particular branch of medicine, but at the present time transfusions are being given daily by house physicians and house surgeons throughout the country. As the resident medical officers of to-day are the practitioners of to-morrow, it follows that transfusions have passed out of the hands of the specialist and can be undertaken by any practitioner familiar with the technique. The methods here described do not attempt to include all the various ways of giving blood, but they do claim to be both simple and efficient.

Blood Groups.

It has been found that blood indiscriminately transferred from donor to recipient may produce in the latter dangerous and even fatal symptoms, so that the suitability of the donor's blood must always be ascertained before commencing a transfusion. These symptoms, which include precordial pain, urticaria, collapse and sometimes death, are due to the fact that in some cases the corpuscles of the donor are agglutinated by the serum of the recipient, and if this takes place that particular donor should on no account be used.

There are four main blood groups, described as 1, 2, 3, 4. Group 1 is known as the universal recipient, as he can receive blood with safety from his own group, and also from Groups 2, 3 and 4. Group 4 is known as the universal donor, as he can give blood to his own group, and also to Groups 1, 2 and 3. All groups may receive blood from similar groups, or from Group 4. Group 4 can only receive blood from Group 4. The most common groups are 2 and 4.

In order to ascertain the group to which any particular individual belongs, it is necessary to procure a small quantity of Group 2 and Group 3 sera ; these are usually supplied ready for use in sealed capillary tubes. The serum is then expressed on to a white porcelain dish—a household plate will serve—previously marked so that the groups will not be confused. A drop of blood of the unknown group is then obtained, and about as much as will fill the eye of a large needle is mixed with the serum. It is a good thing to use two needles, using the eye of one for Group 2 serum and the eye of the other for Group 3, and by this means there is no possibility of mixing even a small quantity of the sera. The dish should be gently agitated so as to mix thoroughly the sera and corpuscles. If agglutination takes place, the appearance is that of a sprinkling of Cayenne pepper ; whilst if there is no agglutination, the two drops remain as they started after the original mixing. A hand lens may be used for observing this, but it is usually unnecessary, agglutination being obvious to the naked eye. If agglutination should occur it will be seen in the first minute or two if fresh active serum is used, and the mixtures need never be watched for longer than five minutes.

It is important to note that if tubes of the known sera be kept for any length of time they should be stored in an ice chest, otherwise they tend to deteriorate, and fallacious readings may result if the tubes have been kept at ordinary room temperature. Reference is then made to the accompanying table, and the group determined.

Corpuscles	Serum			
	1	2	3	4
1	—	+	+	+
2	—	—	+	+
3	—	+	—	+
4	—	—	—	—

For example, if there is agglutination in both drops, then the blood is Group 1 ; if there is no agglutination in the Group 2 serum, but there is in the Group 3, then the blood belongs to Group 2.

If no known sera are available, then the patient and donor cannot be grouped, but a direct agglutination test may be carried out in order to discover whether the particular donor is suitable

for the patient, even though neither blood group may be known. This is performed by taking a little blood from the patient and obtaining the serum from it. This may be done either by withdrawing about 1 c.c. of blood and placing it in a narrow tube, when the serum will gradually separate, or by filling a capillary tube with blood, and after coagulation a string clot can be pulled out, leaving sufficient serum for the test.

The test is carried out in exactly the same manner as described under grouping, the drop of the patient's serum being mixed with the blood of the donor. If no agglutination takes place, then that donor is suitable for the patient.

When giving a transfusion to a patient with one of the "blood diseases" (pernicious anæmia, etc.) it is always safer, even though the groups of both donor and recipient are known, to do a direct agglutination in addition, as in practice these "anæmic" bloods do not always behave as in theory they should do, and agglutination occasionally occurs in groups which should be quite compatible.

The Transfusion (Citrate Method).

The most simple method of all is to administer the blood in exactly the same manner as an intravenous saline is given, and with this method a minimum of apparatus is required.

The essentials are : a flask graduated up to 1,000 c.c., a funnel, some rubber tubing, glass connections, a clip and needles. In order that the blood may not coagulate, it must be received direct into an anticoagulant, and the flask, sterilised either in the autoclave or by boiling, must be prepared in advance. Into the flask is introduced 50-100 c.c. of sterile normal saline at about 100° F., to which is added the necessary quantity of sodium citrate. This quantity naturally varies with the amount of blood it is proposed to take : 1 gm. of citrate should be sufficient for 400 c.c. of blood, though it is probably safer to add rather more than this. The citrate can be obtained from many chemists made up in solution, a sterile ampoule containing 1 gm. of citrate in 3 c.c. The flask, fitted as shown in Fig. 1, should now be corked, and the first stage of the transfusion may be started.

The donor should preferably be a young healthy male with prominent veins, and only those with no taint of venereal disease, malaria, etc., should be used. He should be lying on a couch or bed with the arm selected on the edge. A tourniquet, or, better

still, a sphygmomanometer, should be placed round the arm well above the region of the antecubital fossa, and the armlet inflated until the dial reads about 80 mm. It is of the utmost importance that the arm be not compressed too tightly, or else the blood will not flow, hence the advantage of the sphygmomanometer over the tourniquet. When pressure is put on the arm the veins in the region of the bend of the elbow, the median cephalic and the

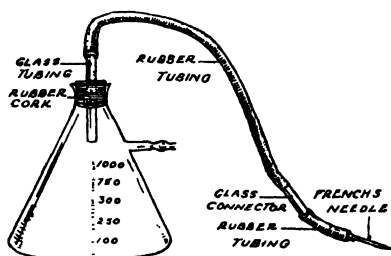


FIG. 1. FLASK FITTED FOR TAKING BLOOD.

median basilic, will become prominent, and can be made even more so by the donor making a fist.

The needle—and the type known as a French's needle is recommended—is now introduced into the vein in an upward direction, and if this manœuvre is performed successfully blood will be seen through the glass connector coming down the tube into the flask. The latter should be held low down, preferably by an assistant, and gently rotated in order to mix the blood and citrate together. As the blood flows the donor should clench and unclench his fist at intervals, about twelve to fourteen times per minute, and this pumps the blood from his vein into the flask. The assistant should be most careful not to put any tension on the tube attached to the flask, as if this occurs the needle may be pulled out of the vein, and the latter rendered useless for that particular transfusion. The blood should flow in a fairly rapid stream, accentuated every time the donor clenches his fist, and unless the blood does flow rapidly there is danger of coagulation between the needle and the flask. If blood ceases to flow, or the rate suddenly decreases, a little gentle manipulation of the needle, such as a very slight withdrawal, will usually start the stream flowing again.

In order to prevent coagulation it has been suggested that sterile paraffin should be run through the tubing before taking blood, but

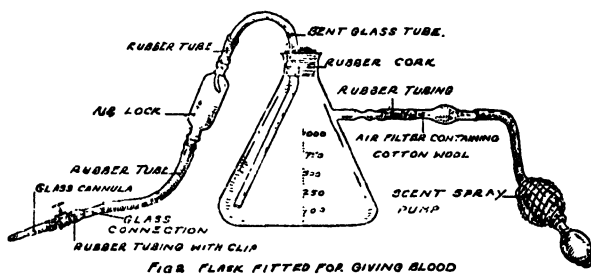
I have never found this procedure necessary. As soon as the requisite amount of blood has been obtained, the armlet is deflated, the sphygmomanometer removed, the needle withdrawn, a wad of sterile wool placed over the puncture, and the donor made to flex his elbow as far as possible. He should then be given half to one pint of hot coffee and made to rest for an hour, after which he may go home. During these attentions to the donor the flask of blood should be placed in a vessel containing warm water, so as the blood will not cool.

Some controversy has taken place as to whether a local anæsthetic should be given to the donor. If the needle is as sharp as it ought to be, this is seldom necessary, and most donors whom I have asked prefer to do without it. One of the objections to a local anæsthetic is that when the vein is not easily seen it may be masked altogether by the injection. Some operators give a small bleb of anæsthetic intradermally, and make a tiny incision in the skin just large enough to take the needle, and this avoids the force sometimes necessary in introducing the needle through the skin. It should never be necessary to cut down upon the vein of the donor, and most bodies supplying donors, such as the London Blood Transfusion Service, rightly insist that this shall not be done. It cannot be emphasised too strongly that the needle should be sharp, and I have seen more than one transfusion ruined as a result of a blunt needle tearing the vein and causing a hæmatoma, which renders the vein useless for some time.

Everything is now ready for the administration of the blood to the recipient. If one of his veins is visible the blood can be given through a needle inserted into the vein, but if the vein cannot be clearly made out, a condition which obtains in many bloodless subjects, it must be dissected out, and a glass cannula inserted. If the intravenous needle is used, a tourniquet may be applied above the vein in order to render it more distinct; as soon as the needle is inserted the tourniquet should be removed. The funnel and rubber tubing, including the needle, should now be filled with warm sterile normal saline until the latter is visible at the lowest part of the funnel, and a clip applied at the needle end. The tubing is then gently massaged in order to expel any air bubbles which will come to the surface of the saline showing in the funnel.

The needle or cannula is now introduced into the vein, and the warmed blood gently poured down the side of the funnel until

the latter is full, the clip released, and the funnel elevated about twenty-four to thirty inches, when the blood flows into the vein by gravity. Towards the end of the transfusion the remaining blood may be somewhat "frothy," and this froth must on no account be introduced into the vein, so that the injection should be stopped by tightening the clip before the froth has left the funnel. An assistant should always be present, as it is most difficult to carry this out single-handed; it is essential for one to keep the needle in the vein of both donor and recipient, as the slightest movement may dislodge it and cause failure.



Within a few hours of transfusion by the citrate method the recipient may have a rigor and high temperature. This is of short duration, and I have never seen harm result, unpleasant though it may be for the patient. It seems to be more common in those cases suffering from a "blood disease" than in the purely surgical cases.

Another method of giving citrated blood is by pumping the blood direct from the flask into which it is received through an air-lock into the vein of the recipient. The apparatus required consists of a stout graduated glass flask, with side-piece, rubber corks, air filter, an air-lock, glass tubing and glass connectors, rubber tubing and clip. In addition, a pump, scent spray type, and an evacuator, are required (Fig. 2).

The blood is taken from the donor in exactly the same way as has already been described. The cork and tubing used during the taking of blood is now removed, and the cork with the bent glass tube, to which is attached the air-lock, tubing and cannula, is inserted in its place.

The tubing is filled with warm sterile saline up to the top of the air-lock, and the clip tightened; this can easily be done by attaching

the evacuator to the air filter and gently squeezing, the needle meanwhile being dipped in the saline solution. This procedure can be done whilst the apparatus is being prepared, as once the clip has been tightened the cork can be removed from the flask, and none of the saline will escape when it is laid aside. The evacuator is now removed, the pump substituted, and all is ready to start. Once the needle or cannula is in the vein, the clip is loosened, and pumping started. As the blood flows over into the air-lock, a certain amount of the saline in the latter is displaced by the air contained in the bent glass tube, but after that the blood level remains about the middle of the air-lock until the transfusion is finished. When the last of the blood is going in, this level in the air-lock begins to fall, and at that point the clip should be tightened and the transfusion ended. In the use of this apparatus there are two practical points which are of importance: firstly, the cork should either be tied on to the flask or held firmly in by an assistant when the blood is being pumped in; and, secondly, it is advisable to secure the rubber tubing to the glass by means of some soft copper wire, as after the tubing has stretched leakage may take place at these joints. The advantages of this method are that it is more easy to work single-handed than the funnel method, and that the blood is never exposed to the air.

Other methods of giving blood usually require more complicated and more expensive apparatus, and are not so suited to the occasional transfusion which may have to be performed by the practitioner. These include the giving of whole blood and defibrinated blood.

Indications.

The indications for blood transfusion are many and varied, but can be divided roughly into surgical and medical.

Surgical.

The chief indication is hæmorrhage, and transfusion may be done either simply to make up for blood lost accidentally, or to enable the surgeon to operate on a bloodless patient. If given for the latter cause, it may be done before, during or after operation, and in very severe cases may be performed in all three periods. There is no doubt that transfusion has enabled surgeons to operate successfully on many cases in which, without it, operative measures would have proved fatal.

Medical.

Pernicious Anæmia.—Before the introduction of liver therapy, transfusion was almost a routine during some period of this disease. At the present time it is almost unnecessary, except in those cases which are moribund when first seen. Such patients are often unable to take food, and an initial transfusion is the most rapid method of restoring them to normal health. Severe anæmias of the pernicious type which do not respond to liver or hog's stomach may be benefited permanently by a transfusion.

Purpura.—When excessive bleeding has taken place, transfusion may be a life-saving measure. It is almost always necessary before splenectomy in those cases of essential thrombocytopenic purpura hæmorrhagica.

Hæmophilia.—Transfusions every few months are said to benefit these cases. It should always be done prior to any possible operative measures.

Leukæmia.—May be done as symptomatic treatment. It never does any lasting good.

Hæmatemesis and Melæna from Peptic Ulcers.—In severe cases transfusion is recommended, even though no surgical interference is anticipated. The idea that the introduction of fluid into the circulation will raise the blood pressure and restart the bleeding is not borne out in practice.

Ulcerative Colitis.—Severe cases are under-nourished and anæmic, and a blood transfusion, repeated if necessary, is often the turning point in their illness, and should always be given if they do not respond readily to the usual treatment.

Hæmorrhage in the Newly Born.—Transfusion often is the only remedy, and should always be done. Blood may be taken (5–10 c.c.) direct from a parent's vein, and injected intramuscularly into the infant, and repeated in a few hours. If this is done rapidly clotting will not occur. Given thus, the blood need not be grouped.

Marasmus.—Good results have been reported from transfusions in this condition, though, personally, I have never seen them.

Finally, as regards the quantity of blood that should be injected: there is no hard-and-fast rule, and it must be left largely to the discretion of the operator. Five hundred c.c. is an average amount for an adult to receive. If it is desired to inject very large quantities, it is better to give repeated transfusions than one enormous one.

CHAPTER XII.

SUDDEN INSANITY

BY

EDWARD MAPOTHER, M.D., F.R.C.P., F.R.C.S.

Medical Superintendent, Maudsley Hospital; Physician for Psychological
Medicine, King's College Hospital.

FOR practical purposes and subject to certain corrections presently to be specified, the phrase "sudden insanity" may be used as a general term denoting a type of crisis which is among the most difficult and distressing of all those with which the family doctor is called on to deal.

Such crises naturally raise questions in the first place of diagnosis in a broad sense (including those of pathology and prognosis), and these are as clearly medical problems as those similarly arising in any other clinical emergency, even though here question and answer may often need for the present to be framed in psychological rather than in physical terms.

But when in connection with sudden insanity we pass to questions of treatment (including placement), we are apt to be confronted with quite special considerations. While here, as always, treatment should primarily depend on pathology and prognosis, the situation is apt to be complicated by the need to consider limitations which depend on law, financial position, and the availability of places of treatment that are suitable and acceptable. In fact, such considerations are usually so dominant in relation to the question of treatment that any discussion on this without regard to non-medical aspects of the problem would be wholly wanting in reality. Some reference must therefore be attempted to both aspects of the problem: the medical, and that which for lack of a better general term may be called administrative.

Before passing to the discussion in any detail of either, I should perhaps admit that a captious critic might be inclined to call my title a journalistic misnomer, sacrificing correctness to conciseness;

possibly, from the standpoint of strict accuracy, "acute mental disorder," or "acute psychosis," would be a better heading. Development within a few days of mental disturbance of a kind entirely incapacitating the patient, and sometimes rendering him a danger to self or others, occurs fairly often. On the other hand, onset of such disorder with an abruptness at all comparable to that of sudden death in an attack of angina pectoris must be extremely rare. Personally, I have never seen such a case, and if restriction in meaning of the word "sudden" were carried to pedantic lengths, then perhaps the size of this article might be shortened to that of the well-known chapter on snakes in Ireland.

An apology for the word "insanity" in my title will also serve to bring out some points that need emphasis. Insanity is, of course, a legal rather than a medical term, implying mental disorder of such kind and degree as to abolish the patient's legal responsibilities, and such as to necessitate depriving him by legal process of ordinary civil rights, *e.g.*, personal liberty and control of property.

A doctor confronted by a case of acute mental disorder (whether as consultant or general practitioner) will usually find himself expected—in the real or supposed interest of the patient, and in deference to the feelings of the relatives—to co-operate in averting and deferring as far as possible acceptance of the legal implications involved in the term insanity. It is often a task of extreme difficulty to reach a definite decision as to whether continuance of treatment is justifiable at home or unofficially at a nursing home. Having reached an adverse decision, one has often a most disagreeable and onerous responsibility to convert reluctant relatives to the same view. Yet, apart from any conscience which one may have about the law, the life of the patient often turns upon correct decision and successful persuasion about this point.

After this preamble, one can pass to consideration of those more purely medical problems which are apt to arise in connection with "sudden insanity," and in the first instance to the question of its ætiology.

One may say at once that principles summarising the manner in which are determined the occurrence and form of mental disorders generally, apply with little modification in the special case of sudden insanity.

Every mental process at any time is necessarily the result of the interaction of factors falling in four groups: (1) Tendencies

inherent in the innate structure of the organism. (2) Tendencies associated with various ages and physiological events and epochs, *e.g.*, those of sex life. (3) Tendencies due to traces of previous mental experience (recent or remote). (4) Tendencies due to traces of physical influences, *i.e.*, acquired modification of reaction—tendencies at the conscious level of the organism by influences other than its own previous activity.

All these factors, which may briefly be summarised as heredity, epochs, experience and physical influences, *must* in varying measure determine the form of any mental reaction. The occurrence of psychosis is a rather different question. Theoretically, an anomaly in one of the above groups of factors might suffice to produce in consciousness and behaviour a divergence beyond normal bounds of such kind, degree and duration as to constitute a psychosis. In actual fact, however, psychosis is generally due to co-operation of factors in more than one of these groups. Occasionally nothing but an inherent tendency can be traced as an explanation. More commonly, even such psychoses as must be termed endogenous are attributable to the combined influence of inheritance and some psychological epoch such as puberty, childbirth or old age. Still more commonly co-operating with factors belonging to one or both of the above endogenous groups is the residual effect of some exceptional mental stress. So long as the traceable causes of a psychosis are limited to factors belonging to one, two or all of the first three groups, so long is the psychosis said to be *functional*.

It is said, on the contrary, to be *organic* if the presence of a physical pathogenic agent can be observed directly, or can be deduced (1) from lasting changes of structure observable during life or after death, or (2) from the presence also of anomalies of reaction (such as fits or paralysis) at levels of the nervous system lower than that of conscious behaviour.

The term "functional" is merely a confession of ignorance. Yet at present it must be admitted that the most careful examination so often fails to identify a physical factor that four-fifths of all cases of psychosis must still be termed functional. Even in the ætiology of the remaining organic fifth it must always be remembered that factors of the functional order are apt to play a part, and that this may even be the dominant one.

So much for the general principles summarising the determination of the *occurrence* of psychoses. Turning now to the

settlement of their *form*, it may be said that the best guide to the understanding of these is to be found, not in formulæ couched in terms of mystic entities like the Unconscious Mind, but rather in conception such as those of Hughlings Jackson and Pavlov, which cover alike the genesis of symptoms in functional and organic psychoses and those in disease of lower levels of the central nervous system. The most useful conception of all psychoses is to regard them as exhibiting in varying measure three main types of symptoms :

(a) *Reductive Symptoms*.—(1) Incapacity to respond selectively to that complex of present stimuli which we call the outer world (imperception and confusion). (2) Incapacity to synthesise present percepts with selective revival of past responses in such a way as would constitute proper discrimination of subjective and objective, and of time, space and other relations, this incapacity showing itself in such defects as amnesia for passing events, apathy and anergia, depersonalisation, disorientation.

(b) *Release Symptoms*.—(1) Decontrol of innate, emotional and instinctive tendencies common to all members of the species, such as joy, grief, fear or anger. This decontrol commonly takes the form of increasing any emotional or instinctive disproportion which habitually prevails in that person. (2) Decontrol of habits and all irrational trends of a more personal kind which have become conditioned in the particular individual.

(c) *Reactive Symptoms*.—Those originating as attempts to hide or compensate the reductive symptoms and their consequences, such compensation, however, being rendered faulty by the existence of those very primary disabilities which it is designed to mask or remedy.

The clinical picture of any psychosis (whatever its ætiology) is apt to comprise symptoms of all three types. Often it also includes several varieties of one type (*e.g.*, at the same time exaggerated liability to depression, fear and anger). In other words, it should be realised that whether in functional or organic psychoses, mental syndromes, such as confusion, depression, anxiety state, hysterical or paranoid reactions, are abstractions of a simplicity which is always far in excess of clinical reality.

The development of a competent psychiatrist consists mainly in the slow evolution of an adequate sense : (1) of the futility of looking for single causes, solitary syndromes, standard courses or

specific cures; (2) of the double futility of trying to propound constant correlations between any two of these abstractions.

Consideration of these general principles governing the determination of occurrence and form of psychoses generally may help to make more intelligible a summary of clinical facts about "sudden insanity." This may occur either as a functional or organic condition, *i.e.*, without or with identifiable physical basis.

Functional Psychoses.

It is perhaps a truism to say that while many cases of psychosis (especially manic and depressive) occur in those whose history shows an inherent and sometimes inherited tendency without any other factor being traceable, yet such attacks are rarely sudden. Abrupt onset seems to require the intervention of some precipitating circumstance, though often stresses will suffice which are the common lot (such as loss of parents).

Influence of Epochs and Events of Sex Life.—It is not very uncommon soon after puberty to find that girls of manic depressive diathesis have at each menstrual period, or at many of them, a transient emotional swing (usually manic) which may reach a point justifying the term "sudden insanity." On a few occasions I have known marriage and its consummation followed, in girls to whom the nature of the latter had been unknown, by a severe breakdown. In two instances I have seen the same result in women follow the persistent but ineffective attempts of a semi-impotent husband. I have only once known diagnosis of illicit pregnancy followed by an acute psychosis, and then ~~it was due to mistaking the nature of an ovarian cyst.~~ On the other hand, the fact of illegitimacy undoubtedly influences the incidence of insanity later in pregnancy. This latter may be very swift in onset, as may be ~~puerperal psychosis~~—especially that occurring immediately after delivery. ~~These insanities of childbirth are usually functional;~~ cases associated either with eclampsia or with sepsis are comparatively rare. The great majority appear to be a resultant of some unknown specific failure of adjustments due at childbirth, with a neuropathic make-up, and often with a current emotional stress, which may be of any kind.

Mental disturbance is common about the climacteric, but such is usually insidious in my experience. I have, however, known at least two instances then of somewhat abrupt psychosis

with delusions of pregnancy. In both cases it was a question of reinstatement years later of a fear of illicit pregnancy which at an earlier age the woman had had good reason to dread.

Traumatic Experience.—More common than psychoses that might reasonably be ascribed (at least in part) to such physical mal-adjustments as endocrine imbalance, are cases following death or insanity of a child or parent or spouse. That such occurrences are common is a matter of plain clinical fact. It is a fact of some importance as establishing that belief in psychogenesis is not mere sentimentality. This latter view formerly prevailed among those whom medical education had left with a simple pride in membership of the "hard-boiled" school, but the number of such is declining. Their *a priori* reasons for scepticism about the pathogenic effect of strong emotion are, of course, quite invalid. Emotion is itself a disturbance of the vegetative, nervous and endocrine systems, and when excessive may leave on these traces which constitute the very essence of a psychosis. A point to be noted is that when the occurrence of overwhelming emotion results eventually in eruption of a psychosis, this usually follows after a period of incubation. It seems to be recurrence in memory rather than occurrence with perception (however intense) that is eventually effective. In the War it was more often in a hospital behind the line than in the trenches that there developed acute psychoses following some experience of peculiar horror. In the trenches perhaps distraction by the demands of the situation protected against persistent brooding.

Form of Acute Psychoses.—It is impossible here to enter upon any detailed discussion of the way in which acute cases of psychosis exemplify the relation of form, on the one hand, to the nature of a traumatic episode; on the other, to the temperament of the individual and his remoter personal experience. A high proportion of cases, of course, take the form of confusion, with clouding of consciousness, incoherence, and amnesia for current events, and often hallucinations. In others, the picture is rather that of overwhelming grief, either of the distracted, agitated type, or in the form of total prostration. A woman who, after being bound over on a mistaken charge of shoplifting, had spent six months preparing a refutation, and was, on reappearance, hustled out of the dock without a hearing, passed into a state of flaccid stupor which for the first week might have been mistaken for concussion. Others, to whom the world

has become abhorrent, display, in the form of mute rigid negativism, a universal resistance to any intrusion by it into consciousness—a sort of hedgehog reaction. A woman who had hitherto suffered in silence the infidelities of her husband passed for weeks into a state of homicidal frenzy when he, having secured leave from the Army on account of her first childbirth, spent the night with one of his mistresses.

In many instances disaster produces a psychosis which is not merely, as in the last case, a reversal of the ordinary disposition, but also the opposite of what the circumstances would reasonably evoke. An extreme instance of this reactionary mania was the death of a man in a padded room after spending the week following his wife's funeral in ceaseless restless activity, combined with laughing, joking, singing and shouting.

Organic Psychoses.

Discussion of sudden breakdown has intentionally been centred mainly upon functional cases. These are not only more frequent, but psychologically at least more interesting. If space permitted it would be easy to demonstrate that temperament and individual experience determine the form and content of organic psychosis as of functional, and that such form and content exemplify processes of distortion which are identical in the two instances. But all this tends to be more obvious in organic mental disorder when it is slowly progressive than when it is acute in onset. In the latter case confusion and incoherence tend to dominate and cloud the picture.

The number of organic conditions which may give rise to sudden insanity is large, but the number of cases due to any one of these cases is small.

Head injury is one of the commonest. A number of the less grave mental disturbances which may result from this are, of course, well known. These include the characteristic retrograde amnesia for a period before the injury, the transient symptoms of cerebral irritation which may follow concussion, neurotic disturbances which are usually called traumatic hysteria, and a generalised organic reduction of function that may go on to dementia. It is sometimes said that with definite head injury associated with fractures of the skull, grosser psychoses are very rare. This is not really the case: I have seen at least a dozen such myself.

In two cases there dated from the injury a second attack similar to one from which recovery had occurred years before this. In these head injury cases the relation of the psychosis to prevailing temperament and to previous emotional experience is often very clear. The prognosis is actually good, even though duration may exceed two years. Finally, occasional paroxysms of extreme violence may be seen in men at other times well-conducted. Possibly they represent an equivalent of traumatic epilepsy, and, like this, they are aliable to be induced by a very small dose of alcohol.

Idiopathic Epilepsy.—Paroxysms like those just mentioned as occurring in traumatic cases occur likewise in the idiopathic form. Such an outburst may occur as an isolated episode in an epileptic who at other times shows practically no mental signs of the disease at all, while in other cases emotional outbursts of some kind may be recurrent and even constant in form. Delusional hostility to one person, often the wife, is fairly frequent in such spells.

General Paralysis.—The first symptoms of this disease to attract attention may be a fulminating psychosis, and this may be precipitated by injury or shock. A well-known London surgeon, whose professional intimates had noted some irresponsibility but who had otherwise passed for normal, had a motor car accident. The next day, lying in bed, he thought he was driving the German Emperor round the room. He died of galloping G.P.I.

Cerebral Tumour.—In a far larger proportion than is commonly realised the first symptoms of cerebral tumours (if located in "silent" areas) are mental. In a few of these cases the psychosis is abrupt, and apparently associated with rapid rise in intracranial tension owing to hæmorrhage into the tumour or surrounding oedema.

Disturbances of Cerebral Circulation.—I have seen several cases of acute psychosis due to cerebral embolism in patients with valvular heart disease, and others coming on abruptly with auricular fibrillation and with paroxysmal tachycardia, and passing off with them.

Toxic Conditions.—Sudden insanity in a patient already under one's treatment should always raise doubts as to whether this may be responsible; the drugs most likely to be so are bromides, belladonna and salicylate. Of all poison which may be responsible for acute psychoses, alcohol is, of course, most frequently so; but this very fact perhaps makes it unnecessary to say more here. Of endogenous toxæmias, those of uræmia and of rapidly progressing

diabetes may induce a sudden mental disorder which usually takes the form of a delirium with hallucinations. It is well to remember that, on the other hand, a very similar condition may result from hypoglycæmia due to overdosage with insulin. Finally, the acuter forms of mental disorder due to infections of all kinds are not only practically indistinguishable from one another in virtue of form alone, but their similarity to those due to non-infective toxæmias is also very close. Of such acute infective psychoses one of the commonest forms in my experience is that following unduly extensive extraction of septic teeth.

Prognosis of Acute Mental Disorders.

It is a general rule in connection with nervous diseases that while form of symptoms is largely determined by the parts affected, the course is determined by the factors interacting in production of the disease, and the form and course are therefore largely independent. This is true of all psychoses, and since "sudden insanity" may be due to such a multitude of causes in different cases, and is usually due to interaction in varying measure of several factors even in one case, it is clearly impossible to lay down any rules of general application save in the vaguest terms.

It is necessary in the first place always to determine how far any organic factor is responsible ; in so far as that is the case, the possibility of removing such factor and its effects dominates prognosis. Apart from this, suddenness of onset itself is probably in functional cases the single factor of greatest weight in justifying a hopeful forecast. It is so because in a general way it betokens that exogenous and probably transient causation plays a large part in the case.

The presumption of recovery will be strengthened by positive knowledge concerning the importance in causation of any mental trauma (such as a love affair) the nature of which suggests the likelihood that this importance will wane. Discussion of the significance of various ages and epochs for prognosis would take too long, but it should be said that attachment of unfavourable significance to history of either family, or personal tendency to psychosis, may be a mistake. If such history be that of a recurrent but recovering condition, the outlook is obviously improved, at least as regards the outcome of the present breakdown.

Treatment.

It has been said, under the heading of prognosis, that the framing of general rules for a condition of such variable causation as "sudden insanity," except in the vaguest terms, is impracticable here. The same applies to what one may call the truly medical aspect of treatment; but something may be said in a summary way about what has been termed the administrative aspect of the matter. No purely legal question arises concerning the legitimacy of treatment of any patient in his own home. Section 315 of the Lunacy Act, however, forbids the treatment for payment anywhere of a certifiably insane person without the authority of the Board of Control—the Board being judge as to certifiability. Probably, however, treatment in a nursing home of a case of recent and acute origin for a month or two would hardly be held illegal, especially if in the causation any organic factor played a considerable part.

Therefore, in the cases now under consideration, the choice as to whether treatment should be in a recognised mental institution or in a nursing home, or the patient's home, is usually one of feasibility and advisability rather than legality. Frequently the relatives are the worst people imaginable to have charge of the patient, and this is often truest when the idea is most forcibly opposed.

Conditions which are specially liable to make institutional treatment imperative are noisiness, extreme restlessness or violence, refusal of food, or pronounced suicidal tendency. Apart from any other considerations, cost tends to be a determining factor. A mental case of such severity as to need special nurses is apt, at least in a London nursing home, to cost twenty guineas per week; and since the main cost is that of nursing, it is not much less at home. Treatment under official auspices having been decided upon, many problems remain, both as to law and the existing provision of suitable institutions. The actual law, as modified by the Mental Treatment Act, 1930, is now as nearly identical for private and rate-aided classes as makes no difference. Both can now be treated as voluntary, temporary, or certified patients. The introduction, however, of temporary treatment, which might have served a useful purpose in discriminating transient cases, has been rendered almost useless, on the one hand, by restriction to patients who are incapable of expressing any wish, and, on the other, by the fact that in effect, and in all but name, it is certification. The

main defect, however, still remains that of institutional provision. For those with means, the position is moderately satisfactory. As far as the poor are concerned, the fact that the passing of the Mental Treatment Act almost coincided with the beginning of "the slump" has delayed all progress. No local authority has yet established, as they might under the new Act, any institution reserved for voluntary cases, as the Maudsley Hospital is in London, or for the treatment of recent cases (whatever their legal status) apart from the chronic. This situation represents a failure to meet demands of the public, which are in some degree reasonable, though possibly based partly on tradition. It may be said that conditions at the public mental hospitals are now so good that medical requirements are largely met for those needing any protracted treatment, and that even the public prejudice (so far as it is based on tradition) is dying down. On the other hand, conditions for the poor during that initial stage which has to be passed in the observation ward before reaching a public mental hospital still leaves a great deal to be desired; and these are the conditions which are apt most immediately to concern those cases of sudden insanity which form the subject of this article.

CHAPTER XIII.

MIGRAINE AND ITS TREATMENT

BY

FERGUS R. FERGUSON, M.D., D.P.H.,
F.R.C.P.

Assistant Physician, Royal Infirmary, Manchester ; Lecturer in Clinical Medicine, Victoria University, Manchester ; Honorary Consulting Physician, Manchester and Salford Royal Hospital for Skin Diseases.

So many medical men suffer themselves so much at the hands of migraine, that surely, apart from a consideration of our patients, we cannot feel that we are wasting our time in reviewing the present position of our knowledge of this condition. Although at times a trivial complaint, yet it can constitute one of most the disabling of common maladies. It seems difficult at first to realise that "a mild attack of the megrims" is but a variety of a disease which at other times can incapacitate a patient for weeks on end or even prove fatal. Despite a vast amount of interest in the subject, we are still uncertain about its definition, its pathology, its nature, and its cure. Migraine has been the happy hunting ground of the theorist, and the problem has been attacked by representatives of all branches of medicine. Each in turn has discovered in migraine phenomena pertaining to his own speciality ; each in turn has hit upon the true nature of the malady ; in turn each has found the infallible remedy. Such mutually exclusive viewpoints cannot all be entirely correct, and the multiplicity and diversity of the hypotheses suggest that the problem is much more complex than was imagined at one time. One of the usual errors in describing and explaining migraine is to ignore the less typical though common forms which the malady may offer. Herein lies our first difficulty, for at the outset we must agree on what constitutes migraine, before we can discuss its treatment.

What is Migraine ?

In the text-books migraine is usually described as a paroxysmal affection comprising severe unilateral headache preceded by visual

phenomena, and followed by nausea and vomiting. Undoubtedly this account is sufficiently accurate to enable a large number of cases to be recognised, but it gives no hint of the occurrence of a far greater number of slighter and incomplete forms, nor of the rarer severe and even catastrophic manifestations. The following definition of migraine, and the conclusions which are put forward as to ætiology and treatment, are based on an investigation undertaken in conjunction with Dr. Macdonald Critchley of over 300 cases. Detailed study of migrainous subjects reveals quite clearly and definitely that attacks may differ extraordinarily in degree and complexity in the *same* person; and with advancing years and with varying circumstances the attacks may alter and cease to correspond with the classical description of the disease. Nevertheless, these slighter and less common types of attacks must surely be merely variants of "ordinary migraine."

Acting upon such clinical data, we may state that *any one of the classical symptoms* (teichopsia, headache and vomiting) *may be absent*. Migraine without visual phenomena is recognised by many, but it is doubtful whether the frequency of such is appreciated. Nausea and vomiting are often absent, and in a few instances headache itself may be in abeyance. This is admittedly rare, and might legitimately cause diagnostic argument were we not mindful of the frequent absence of headache in the elderly migrainous subject. Under treatment, moreover, the headache may be cured, but not the other phenomena.

Advancing our argument further, we may state that *any two of the classical symptoms may be absent* and yet the diagnosis of migraine remains. Thus, visual phenomena and vomiting may both be absent, the migraine attack being represented by headache alone. Cases of this type are, indeed, common. It may be asked how in such circumstances the diagnosis is made. The periodicity of the attacks, the character of the headache itself, and some of the accompanying disorders, such as confusion, irritability, anorexia, giddiness, photophobia, and inability to "carry on," are features which distinguish it from other types of headache. A headache which is present on rising (frequently when the patient has felt unusually well on the previous day), or one which leads to drowsiness and sleep, is suggestive of migraine. The very occasional occurrence of other phenomena—teichopsia or vomiting—during one of the patient's "usual headaches" also indicates the migrainous

nature. One may summarise by stating that migraine is the commonest cause of severe periodic headaches, accompanied by prostration, especially if present in the early morning on waking. Bouts of vomiting alone may occur in younger subjects prior to the development of the complete forms of migraine, and also in the migrainous subject during pregnancy. For these and other reasons we affirm that attacks of vomiting alone may constitute a *forme fruste* of a migraine attack.

Some Special Clinical Points and Features.

The headache is often not unilateral.—Although this statement challenges the very name of the malady (hemicrania), nevertheless, careful investigation reveals that in a high proportion (perhaps even 50 per cent.) the pain does not strictly concern one-half of the head. The pain may be more intense on one side, without, however, any strict unilaterality. Pain may be noted in the frontal, parietal, or occipital regions; it may affect the summit of the skull; may occur in the temples, or may be referred to one or both orbits. The location of the pain often varies from one attack to another in the same subject.

In connection with the ætiology, it is of interest to recollect that *migraine headaches usually cease during pregnancy*, and one often hears it said that *migraine ceases with the climacteric*. It must be remembered, however, that the migrainous patient usually suffers an exacerbation at the menopause, and many continue to suffer alarming attacks for some time after cessation of the menses; nevertheless, in the majority of cases, within five to seven years of this date the migraine will have become much milder or have almost disappeared. One is frequently consulted by a life-long migraine sufferer (who has hitherto treated the attacks herself) because she is alarmed, on reaching the age of fifty or thereabouts, by the alteration in type and severity of the headaches.

The analogy between migraine and epilepsy has often been made; the many points of contact between the two disorders serve as useful guides in clinical description as well as suggestive features in ætiology. Just as we arbitrarily describe a major and a minor form of epilepsy, so we may isolate a variety of migraine which, from its severity and at times almost dramatic character, may be spoken of as "migraine major." In this category belong the particularly severe examples of the bilious and the cerebral

types, when the intensity and protraction of the symptoms may arouse doubt as to the original diagnosis. Differentiation will have to be made in such cases from acute abdominal emergencies, gastric influenza, acute food poisoning, on the one hand, and gross intracranial disease, especially spontaneous subarachnoid hæmorrhage resulting from rupture of a cerebral aneurysm, on the other. The accompanying prostration and loss of weight take these examples of migraine out of the category of a minor malady.

We must mention here those cases where neurological symptoms are present in a striking degree. Thus, mental confusion, the paraphasic speech defects, the weakness of a limb or limbs, the feelings of numbness in the face or extremities, may be so marked as to suggest a cerebral tumour or thrombosis. Although these features are not rare even in minor forms, they may occasion difficulty in diagnosis when severe or protracted. To the group of migraine major also belong certain complicating events of a severe and even alarming nature. Not uncommonly a patient may emerge from a severe bout of migraine only to commence immediately another turn. In this way one attack may succeed another for as long as a month. Such cases generally give rise to considerable anxiety and difficulty in diagnosis; upon the analogy of the epileptic phenomenon the term "status hemicranicus" is applied. During this state the patient is never entirely free from headache, though it may fluctuate. Extreme physical and mental prostration may result; the patient may lose a stone or two in weight, and prolonged convalescence may be required. If subjective sensory disturbances occur during each attack, then objective sensory impairment of a hemiplegic distribution may be demonstrable. The relationship with epilepsy is interesting. Epilepsy may occur among the relatives of a migrainous subject; or epileptic and migrainous attacks may alternate in the same person; or epilepsy may cease and be replaced by attacks of migraine, the fits reappearing in later life when the migraine has abated. The term "migraine epilepsy" is best restricted, however, to those cases where unconsciousness—with or without convulsions—occurs at the height of the migraine attack. It is noticeable that when a migrainous attack culminates in unconsciousness the headache is thereby entirely relieved and the attack cut short.

Sometimes the attack of migraine is accompanied and followed for some days by severe neuralgic pain in a limited area of the

face or head. The patient may complain of severe pain localised to the eyeballs, the inner canthus, the side of the nose, temple, malar ridge or mastoid region. Even the neck, shoulders and upper dorsal region may be affected. The pain may commence in the attack, but persist for days or weeks after its finish. This is referred to as "migrainous neuralgia," and in some of these cases careful investigation will reveal a condition of non-articular rheumatism, as betrayed by a tender nodule or thickening in the muscles or aponeurosis of the head and face.

Persistent paralytic manifestations constitute an interesting, though fortunately fairly uncommon, series of phenomena. During the attack of migraine certain neurological signs appear, and though becoming less noticeable when the attack has ended, they may not entirely disappear with succeeding turns, and eventually they become permanently installed. Pupillary inequality may be quoted as an example: during an attack the patient's friends may notice that one pupil becomes much smaller than the other—in the course of time the anisocoria becomes permanent, and the patient may present a small inactive pupil on one side and abnormal, mid-dilated pupil on the other. Persistent hemianopia is another well-known sequel of chronic migraine, but probably the most characteristic is the well-known "ophthalmoplegic migraine," where a recurrent ocular paresis eventually becomes permanent. The third nerve is the one implicated in by far the greatest number of cases, but exceptionally the fourth or the sixth nerves may be concerned. Persistent hemiplegia, with or without aphasia and agraphia, has not infrequently been seen after chronic migraine.

The analogy with epilepsy may be pursued further, and the term "migraine equivalents" may be applied to those episodic events which at times seem to replace a true attack of migraine. Their nature is varied: they may comprise a period of mental confusion and sense of unreality lasting for a few hours; a spell of giddiness or nausea; paroxysmal tachycardia or tinnitus, and, lastly, transient psychoses.

Ætiological and Therapeutic Considerations.

There is little doubt that migraine constitutes an extremely complex problem, and that there are many different factors—external and constitutional—at work in the production of this very distressing malady. There are many different groups and

clinical types, and it will be convenient to refer at this point to the more important of these. The treatment will naturally vary with the suspected causation, and therefore at the end of each paragraph on a particular type a brief outline of the therapy which has been advocated as most likely to benefit that type will be given.

Alimentary Type.

Particular attention has been paid by the French authors to the "bilious," "abdominal" or "duodenal" type of migraine, in which it is thought that the causative factor is an abnormality in the gastro-intestinal tract or liver. The gall-bladder has frequently been blamed for these attacks, but the results of cholecystectomy have in many instances been disappointing. It would seem more probable that the initial cause is a liver dysfunction following a subacute hepatitis, which in turn has resulted from absorption of toxins or bacteria from the intestinal tract or other area of infection. Such hepatitis may allow the passage of organisms into the gall-bladder, causing a chronic cholecystitis or the formation of gall-stones, and may be unaffected by the removal of the diseased gall-bladder unless the source of infection is also attacked. The treatment recommended in these cases is glucose, $\frac{1}{2}$ oz. twice daily, and Decholin tablets, one, three times daily, with instructions to double the dose if the patient feels an attack is impending. There is considerable difference of opinion as to the part played by the "liver-gall-bladder-gastro-duodenal" factor in migraine, but it would appear certain that although its importance has been exaggerated by some writers, yet there are many cases which are intimately associated with hepatic dysfunction and relieved by its correction.

Dietetic Type.

In close association with the above group is the dietetic type; but it is improbable that diet plays an important part in the production of migraine. All manner and varieties of diets have been recommended from time to time—a high-fat or a low-fat diet, withdrawal of all forms of animal protein from the food, a full carbohydrate diet or one with carbohydrates eliminated—each advocate of a particular diet extolling its virtues and turning a deaf ear to the claims of its rivals. If in a particular case it seems probable from a careful history that a particular article in the diet

is an important predisposing factor, or a positive result is obtained from sensitisation tests as for asthma, then it is clear that the offending article must be excluded. Otherwise, in such a protean disease, in which so many diets have been recommended on a purely empirical basis, it seems unwise to prescribe any special diet. It is probable that quantity and regularity are more important factors than quality.

Metabolic Type.

The frequent onset of migraine in the early morning, the occurrence of attacks in many cases after prolonged physical exertion or after starvation, the observations of low blood-sugar readings in several cases, and the relief, especially in children, by the administration of glucose, have suggested that transitory hypoglycæmia may be an important factor. In several cases definite relief seems to have been obtained by the ingestion of glucose as soon as notice of an attack is given, although in other cases an attack has apparently been precipitated by the taking of sugar or sweet foods. It is difficult to correlate these views and findings. •

The observation that acetone is frequently present in the breath before an attack has led to treatment by the withdrawal of fats and giving glucose and alkalis. The suggestion that migraine is due to an inherited or acquired defect in protein metabolism—an inability to detoxicate putrefactive substances derived from animal protein in the intestinal canal—has led to treatment by withdrawal of proteins from the diet. Abnormalities in the uric acid metabolism are invoked to explain other cases of migraine ; or, again, from the association of migraine with chronic nephritis, the view has been promulgated that there is an accumulation in the blood of some toxic substance requiring elimination. It is extremely difficult to assess the importance of the metabolic factor in migraine ; but in obscure and therapeutically refractory cases, where investigation reveals a metabolic error, the appropriate treatment should be instituted.

An Allergic Type.

Some regard the fundamental cause of migraine as an anaphylactic shock. The well-known relationship with asthma, urticaria and angioneurotic oedema supports this view, and has led to the administration of weekly injections of 1 c.c. of horse serum for two months, or to the taking of 0.5 gm. of peptone before each

meal, or to injecting peptone subcutaneously. In some cases the migraine attack is found to occur only after the ingestion of a particular article of food, *e.g.*, chocolate or beans. Thus the treatment of migraine due to food allergy is carried out by "elimination dieting," modified according to the suspected factor.

Vasomotor and Sympathetic Types.

Allied to the above is the vasomotor type, in which it is held that the migraine is the result of a localised intracranial oedema. Erythromelalgia, attacks of angioneurotic oedema, orthostatic albuminuria and syncopal attacks were all present in association with typical migraine in a patient recently under observation. Treatment with full doses of calcium lactate or calcium subcutaneously produces good results in such cases.

At this point it will be convenient to refer briefly to the sympathetic type. Acting upon the supposition that migraine was dependent upon an excessive stimulation of the sympathetic and a resultant spastic contraction of the cerebral arterioles with subsequent relaxation, an attempt was made to control such vasospasticity by the use of a sympathetic depressor—ergotamine tartrate (2 mg. by mouth)—given in conjunction with irradiated ergosterol and calcium; very good results have accrued from this treatment. Dandy has attempted to achieve the same object surgically by removal of the first thoracic sympathetic and inferior cervical ganglia. The results were encouraging, but he preferred to reserve his opinion as to the absolute value of the procedure.

Ocular Type.

There is still much difference of opinion as to the importance of the rôle of errors of refraction and slight ocular muscle balance defects. It is suggested that eyestrain increases the irritability of the central nervous system, and thus produces migraine in a suitable subject. Although there are undoubtedly many cases in which the visual apparatus is primarily at fault and in which migraine is cured by correction of refractive errors, etc., yet one feels that the importance of this factor has been overestimated. Whatever the relative importance of the part played by the eyes, it is obviously our duty to see that no person is allowed to remain for years an unrelieved migrainous subject without very careful ocular testing.

Sinus and Fibrositic Types.

There are a certain number of true migraine cases with well-marked sinus disease in which the treatment of the sinus lesion has led to cure of the migraine. The association of migraine, migrainous neuralgia with non-articular rheumatism, brachial neuritis and scalp tenderness in an attack, separates off a further group—the fibrositic type. These cases should be treated by salicylates, physiotherapy (ionisation), or the local injection of Impletol (a complex combination of a local anæsthetic with caffeine prepared by Bayer). The true cases of migrainous neuralgia respond to alcoholic injection of the Gasserian ganglion or its branches.

Para-epileptic and Cerebral Types.

The similarity in many respects between migraine and epilepsy and their occurrence in the same patient, has led to the treatment of the para-epileptic type by bromides, luminal and the ketogenic diet. Certain cases of typical migraine result from macroscopic intracranial lesions, constituting what might be conveniently referred to as the cerebral type. Tumours in the occipital and parietal regions or ruptured congenital aneurysms have been found in fatal cases of typical migraine.

Endocrine Type.

Almost all the endocrine organs have been blamed at one time or another for attacks of migraine; and as a result X-ray treatment of the thyroid, the use of Lugol's iodine, thyroid extract, adrenalin or whole gland pituitary extract, have been recommended. On the grounds that many cases of migraine, especially those occurring at the menstrual periods, are due primarily to ovarian dysfunction, Collip's placental hormone—Emmenin complex (Glaxo)— $\frac{1}{2}$ to 1 drachm twice daily, except during the menstrual period, has been administered with excellent results. In such cases it is held that the ovarian follicular hormone causes a diminution in size and activity of the pituitary, the swelling of which at the menstrual period would otherwise cause headache by pressure against an abnormally ossified sella turcica or an usually well-developed diaphragma sellæ. Fig. 1 shows a normal sella turcica, and in Fig. 2 is seen a good example of the "bridged-in sella" resulting from abnormally long clinoid processes or ossification of the inter-clinoid ligaments. There is no doubt about the therapeutic value

of an active ovarian follicular hormone in these cases ; but there is much to be said against the view that the headache is due to pressure of the swollen pituitary body against the sella turcica or diaphragma sellæ, for there is ample space for the pituitary body to enlarge laterally. In these cases of intermittent headache in women, Thomson recommended that three injections of theelin, 1 c.c. (fifty rat-units each), should be given in the week before the period is due, but when the headaches occurred at irregular intervals as well as at menstruation, two to three injections of fifty units should be given regularly each week.

Psychological Type.

Speaking generally, there is no doubt about the importance of the psychological factor in migraine, and the association is clear in many cases with mental overwork, anxiety, or "suppressed rage and humiliation." There are, however, in addition, cases due to a specific psychological cause, and in such patients psychotherapy has led to cure.

Treatment.

Success in treatment will depend on the ability to place a particular case of migraine in its proper category and apply the appropriate remedy. For this purpose the complete investigation of a patient with migraine is necessarily extensive, and will include a careful history, physical, psychological, ocular and cerebrospinal fluid examinations, and radiographic and biochemical investigations.

It is customary to discuss the treatment of migraine under two headings : (a) in the attack ; and (b) between the attacks. In the former it is useless trying to "fight off an attack," and we would recommend retirement to a quiet darkened room as soon as possible, and the administration of one of the following : (1) Veramon, gr. vi, repeat in half an hour ; (2) phenacetin, gr. xv, and caffeine cit., gr. v, to be repeated, if necessary, at two-hourly intervals for three doses ; (3) combral, three tablets every six hours.

Between the attacks some of the cases may suitably be treated with glucose, bile acids, theelin, thyroid, special diet, or psychotherapy, according to the special type. The remaining group of migraine cases, which we are unable to place in a particular category, must be regarded for the present as idiopathic, and treated



FIG. 1 Radiographic appearance of the sella turcica in a normal subject.

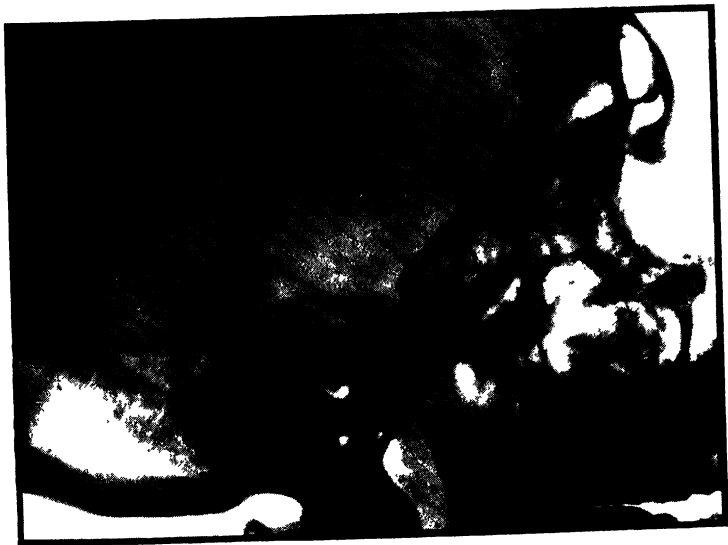


FIG. 2. -A very good example of a "bridged-in sella."

"MIGRAINE AND ITS TREATMENT," by Fergus R. Ferguson, M.D., D.P.H.,
F.R.C.P.

with one of the following : (1) Luminal, gr. $\frac{1}{2}$ -1, *bis die* ; (2) a prescription similar to Gowers' mixture :

R	Sod. brom.	gr. x
	Liq. trinitrin.	m. i
	Liq. strych.	m. v
	Ac. hydrochlor. dil.	m. x
	Tinct. gelsem.	m. v
	Aq. chlorof. ad	$\frac{1}{2}$ oz.
	$\frac{1}{2}$ oz. ter in die	post cibos.

Or, R Ext. thyroid, gr. $\frac{1}{2}$ three times daily.

For the extremely severe cases which do not respond to any of the above measures we would recommend a right subtemporal decompression.

Conclusions.

Migraine is obviously a complicated, interesting and difficult problem, and despite an immense amount of research the essential causation still remains obscure. It seems certain, however, that there must be at least two factors—"central" and "peripheral." In the present state of our knowledge any view must be speculative, but one is inclined to regard migraine as a vaso-motor manifestation associated with spasm of the cerebral arteries—most commonly branches of the internal carotid—due to stimulation of sympathetic fibres, either as the result of direct pressure or *via* the endocrine system, occurring in a susceptible person with the migrainous constitution and with precipitating factors—metabolic, alimentary, ocular, sinus, etc.

CHAPTER XIV.

VERTIGO AND ITS TREATMENT

BY

W. RUSSELL BRAIN, D.M., F.R.C.P.

Assistant Physician to the London Hospital and the Royal London Ophthalmic Hospital; Physician to the Hospital for Epilepsy and Paralysis, Maida Vale, London.

The Nature of Vertigo.

THE name "vertigo" implies that the sufferer experiences a sense of rotation, either of himself or of his surroundings. For many purposes, however, a wider conception is more useful, and vertigo may be briefly defined as a sense of instability, which only in certain forms is associated with distressing hallucinations of movement such as merit the name "vertigo." "Instability" by derivation implies a disturbance of standing, and to understand the nature of vertigo we must briefly review the way in which the normal stability or equilibrium of the body is maintained and modified. The position of the body in space is orientated, for the most part reflexly, under the influence of various afferent impulses. These are derived from:

(1) *The Retina*.—It is clear that vision is of great importance to man in the regulation of his posture. An airman, for example, flying in a fog may fly upside-down without being directly aware of the fact.

(2) *The Ocular Muscles*.—Afferent impulses from the proprioceptors of the ocular muscles play an important part in our perception of visual space; for example, the degree of accommodation and convergence required to fix a sharp image of an object on the macula conveys information as to the distance of that object from the eyes, and the degree of contraction and relaxation of the various extrinsic ocular muscles contributes to our consciousness of "above" and "below," "to the right of," and "to the left of."

(3) *The Labyrinths*.—These are extremely important end-organs in the regulation of the position of the body in space. The three

semi-circular canals, arranged roughly in three planes at right angles to each other, are stimulated by movements of the head in space, while the otolith organs, consisting of calcareous granules suspended among sensitive hairs, appear to be the receptors concerned in appreciating the position of the head with reference to gravity.

(4) *The Cervical Spine*.—The importance of the cervical spine in regulating the posture of the body lies in the fact that it relates the head to the body. Information derived from the labyrinths with regard to the position of the head in space would be of little value if the position of the head could not be correlated with that of the body. This important function is carried out by the end-organs of the joints and muscles of the cervical spine.

(5) Similar end-organs in the lower regions of the spine, the joints and muscles of the lower limbs, and the skin and deeper tissues of the soles, convey information concerning the posture of the trunk and lower limbs in such activities as sitting, standing and walking.

Through these five channels, therefore, we have incoming streams of information concerning the position of objects in space, the position of the body with reference to external objects, and of the different parts of the body relatively to each other. This information must be centrally co-ordinated in order that appropriate reactions may be evoked and carried out by postural contractions and voluntary movements of the bodily musculature. This reflex co-ordination is effected chiefly by the vestibular nuclei, the posterior longitudinal bundle, the nuclei of the third, fourth and sixth cranial nerves, and the cerebellum; while higher levels of the nervous system, especially probably the parietal cortex, are concerned in the perception of space and the conscious orientation of the body.

Vertigo may occur as a result of a deficit or conflict of the information derived from the various sources enumerated, with regard to the position of the body in space, or from a disturbance of the central mechanisms by which the information is co-ordinated and effectively utilised. Vertigo is thus a symptom which can be produced experimentally, and is also a symptom of a large variety of pathological states. Whatever its cause, vertigo of sufficient severity is usually accompanied by certain associated disturbances. When the patient suffers from hallucination of

movement in a certain direction, he is apt to manifest compensatory movements which are reflex attempts to counteract the supposed movement. Such movements in the eyes consist of conjugate deviation and nystagmus, in the limbs of pass-pointing, in the body as a whole of falling; and visceral disturbances, such as pallor, sweating, alterations in pulse rate and blood pressure, nausea and vomiting, occur in severe cases.

Causes and Treatment of Vertigo.

(1) *Vertigo of Ocular Origin.*—The commonest ocular disorder responsible for vertigo is paresis of an ocular muscle which leads to diplopia. The diplopia is due to the false projection of the visual field of the affected eye, and the conflict between the false and the normal projections of space leads to vertigo, which is immediately relieved by obscuring the vision of one eye, preferably the affected one. This is most conveniently done by giving the patient spectacles with a frosted glass in front of the eye.

(2) *Vertigo of Labyrinthine Origin.* (i) *Experimental Aural Vertigo.*—Vertigo can readily be produced by stimulating the labyrinth, which is usually done either by irrigating one external ear with hot or cold water, or by rotating the patient. The essence of these tests is that by artificial means a current is set up in the endolymph such as is normally excited by movement of the head. In the test, however, this occurs when the patient is sitting in a chair or lying in bed. There is thus a conflict between the information artificially evoked from the labyrinth and that derived from the proprioceptors of the limbs and trunk, and giddiness results. Vertigo and the associated disturbances, nystagmus and pass-pointing, are thus utilised to investigate the excitability of the labyrinth and the conductivity of the afferent paths concerned in equilibrium.

(ii) *Pathological Aural Vertigo.*—Aural vertigo of pathological origin is sometimes known as Ménière's syndrome, though the cause to which Ménière attributed the symptom in his cases, namely, hæmorrhage into the internal ear, is a rare one. Pathological aural vertigo may be produced by a number of different disorders. In almost all cases the lesion of the ear involves the auditory as well as the vestibular functions. As a rule, therefore, aural vertigo is associated with tinnitus and deafness. The vertigo consists of a sense of rotation, either of the patient or of his surroundings, or

of both. The most important causes of aural vertigo are : (a) Wax in the external auditory meatus. This is readily diagnosed by inspection, and the vertigo is cured by removal of the wax. (b) Acute otitis media. Acute otitis media may sometimes cause giddiness, but this symptom is frequently absent. (c) Lesions of the internal ear itself are the causes of the most severe and intractable forms of vertigo.

Acute Labyrinthitis.—This is not very common, and is most often due to an extension of a purulent infection from chronic suppurative otitis media. There is also a rare, spontaneous, acute lesion of the labyrinth known as acute serous labyrinthitis. In both, deafness, usually rapidly progressive, is associated with the symptoms of acute vestibular disturbance, vertigo, with nausea and vomiting, nystagmus, forced movements, and ataxia of the limbs on the affected side. There is also pain in the ear, and often considerable pyrexia. The symptoms are those of irritation of the affected labyrinth. The patient tends to lie on the sound side, and pass-points and falls to the side of the lesion. Nystagmus is rotatory, and the quick phase is to the opposite side, to which the patient feels he is falling.

Recurrent Aural Vertigo.—This is a well-defined syndrome which may be produced in several ways. Nasal obstruction and infection and blockage of the Eustachian tube are the commonest predisposing causes of the disorder, which is then probably due to a chronic low-grade infection of the internal ear. In other cases the focus of infection exists in the teeth. Recurrent aural vertigo occasionally occurs in patients with chronic suppurative otitis media. Whatever the cause, the patient suffers from recurrent attacks of vertigo associated with tinnitus and progressive deafness. These disturbances of cochlear function are sometimes unilateral, but are not uncommonly bilateral, though the deafness is often more severe in one ear than in the other. The attacks of vertigo may be mild or extremely severe. When severe, the patient may fall as if shot, usually to the side of the affected ear, and sometimes with sufficient violence to fracture ribs. Consciousness is almost invariably retained during the attack, but exceptionally the disorder of orientation may be so severe that the patient temporarily becomes unconscious. The attack of vertigo may last minutes or hours, and when severe is associated with violent vomiting, precipitated by the slightest movement. When the

patient has suffered from such attacks for some time he often experiences a persistent sense of instability in standing and walking in the intervals between the attacks, and, not unnaturally, he tends to become apprehensive and anxious. Recurrent aural vertigo is a self-limited disorder in the sense that the attacks are likely to cease when the labyrinth is finally destroyed, an issue, however, which may not eventuate for a number of years, during which the patient is exposed to much misery and some danger.

(iii) *Fistula of the External Semicircular Canal*.—Fistula of the external semicircular canal, which is a complication of chronic suppurative otitis media, leads to a distinctive form of vertigo, detected by the fact that intense vertigo and nystagmus are produced by altering the pressure in the external auditory meatus, for example, by inserting a finger.

In addition to the lesions already described, other disturbances of the internal ear may lead to vertigo, for example, otosclerosis, and the action of drugs, especially quinine and the salicylates. Circulatory disturbances also often cause vertigo, though it may be difficult to decide whether it is due to a disturbance of blood supply to the internal ear or to the centres in the nervous system concerned in equilibration. Abnormalities of the circulation are probably responsible for the vertigo not uncommonly associated with athetosis, hyperpiesia, vasomotor instability, severe anæmia, migraine, and increased intracranial pressure.

Treatment of Labyrinthine Vertigo.

During an attack of vertigo the patient should be kept at rest in the posture in which the giddiness is least severe. Sedative drugs will be required, and of these luminal appears to be the most effective. It may be given in one-grain doses by the mouth; but if vomiting is severe, two to three grains of luminal sodium in 20 per cent. solution should be given subcutaneously. In very severe cases morphia may be needed. In acute suppurative labyrinthitis surgical treatment is likely to be required, but the acute serous form should be treated medically along the lines indicated, until the vertigo subsides.

In recurrent aural vertigo any local abnormality in the nose and ears should receive attention. The Eustachian tubes may require catheterisation, and dental sepsis, if present, must be treated. Most patients derive considerable benefit from a prolonged course

of luminal in doses of half a grain, two or three times a day. Quinine in small doses is also advocated. Many patients will respond satisfactorily to these measures, and can be kept in fair comfort until in the course of time the attacks of giddiness cease, often with an increase of the deafness. When, however, the vertigo proves intractable, and especially if it interferes with occupation, surgical treatment should be considered. When deafness and tinnitus are present in both ears it is not always easy to decide which ear is responsible for the vertigo. As a rule this is the ear in which the deafness is most pronounced. An attempt may be made to abolish the functions of the internal ear by the injection of alcohol into the external semi-circular canal, but division of the eighth nerve within the posterior fossa, though a more severe operation, has given very good results, and patients previously incapacitated for work have been able to return to their employment after this procedure.*

(3) *Seasickness*.—Vertigo is a common and troublesome symptom in seasickness, which appears to be due to a combination of disturbances of the labyrinth and of the perception of visual space. The movements of a ship in a rough sea cause an irregular excitation of the vertical semi-circular canals, which leads to a disorder of equilibrium until the subject has adapted himself to it. At the same time his appreciation of visual space is disordered by the movement of the ship relative to himself, and of both in relation to the horizon, and by the occurrence of ocular imbalance, which is probably secondary to the disorganisation of the labyrinthine impulses.

Susceptible individuals should take half a grain of luminal twice or three times a day as a vestibular sedative for two or three days before embarking, and should continue this treatment for the first few days of the voyage. At the onset of the first symptoms the same dose should be given hourly for four hours, and the daily dose may then be increased to two or three grains for several days. When the ship is pitching, the patient should lie athwart the vessel; when it is rolling, he should lie fore and aft. Closure of the eyes, wearing coloured glasses, or bandaging one eye, may be helpful. The diet for a day or two before going on board and during the early days of the voyage should be light and digestible. Plenty of sugar

* "Aural Vertigo: Treatment by Division of Eighth Nerve." H. Cairns and W. R. Brain. *Lancet*, 1933, i, 946.

should be taken to diminish the tendency to ketosis, and an alkaline mixture may be given with the same object ; since constipation is apt to be troublesome, this may conveniently take the form of an effervescent saline purgative.

(4) *Vertigo due to Lesions of the Vestibular Nerve.*—A lesion of the vestibular nerve between the internal auditory meatus and the pons may cause vertigo by irritating the afferent fibres from the labyrinth. Interference with the functions of the cochlear fibres is likely to occur at the same time, causing, as in the case of aural vertigo, tinnitus and deafness. The two most important lesions of the eighth nerve are a tumour occurring upon the nerve, a neurofibroma, and involvement of the nerve in inflammation in meningo-vascular syphilis. Auditory nerve tumour occurs in adult life, and in the early stages may be mistaken for a lesion of the internal ear. It is distinguished from this, however, by the fact that being a progressive intracranial lesion it causes symptoms of increased intracranial pressure, especially headache and, later, papilloedema, and also symptoms due to compression of neighbouring parts of the brain, for example, nystagmus and inco-ordination due to compression of the cerebellum, facial paræsthesiæ and sensory loss, external rectus paresis and facial paresis, from involvement of the fifth, sixth and seventh cranial nerves respectively. When the eighth nerve is involved in meningo-vascular syphilis, other symptoms of this disorder are likely to be present, especially Argyll Robertson pupils ; the Wassermann reaction is likely to be positive, both in the blood and in the cerebro-spinal fluid. Treatment of this form of vertigo is that of the causal condition. Luminal may be used to give symptomatic relief.

(5) *Vertigo due to Defective Postural Sensibility.*—Patients suffering from disorders causing an impairment of appreciation of posture in the lower limbs—for example, tabes dorsalis and subacute combined degeneration—may complain of giddiness, which is most marked on standing with the eyes closed, and is associated with evident instability in these circumstances—Romberg's sign. In addition to treatment of the causal condition, re-educational exercises on the lines suggested by Fraenkel may render these patients more comfortable.

(6) *Vertigo of Central Origin.*—Vertigo may be due to lesions or disturbances of functions of the central paths concerned in the regulation of posture of the body, either in the brain stem, cere-

bellum or cerebral hemispheres. The following pathological states require special consideration :—

(i) *Intracranial Neoplasm*.—Vertigo is a common symptom of intracranial neoplasm, and may occur in association with a neoplasm in any situation, though it is certainly most common in the case of tumours of the cerebellum and brain-stem. There is usually no difficulty in diagnosis in these cases, since symptoms of increased intracranial pressure and of a progressive focal intracranial lesion are usually prominent.

(ii) *Disseminated Sclerosis*.—In this disease, also, vertigo frequently occurs, and is occasionally the presenting symptom. It tends to be transient, but recurrent, and transient recurrent vertigo in a young adult in the absence of an aural lesion should always suggest this possibility. Other signs which may be present in such cases include pallor of the optic discs, nystagmus, intention tremor, diminished or absent abdominal reflexes, exaggerated tendon reflexes and extensor plantar responses.

(iii) *Cerebral Contusion*.—Vertigo is a common symptom in those who have sustained contusion of the brain following a head injury. In such patients it is usually paroxysmal and associated with paroxysmal headache, both symptoms being usually precipitated or exacerbated by excitement, exertion and stooping. Such patients usually benefit from luminal, and tend gradually to recover from their giddiness.

(iv) *Circulatory Disturbances*.—Vertigo is often complained of by elderly patients suffering from arterial atheroma and high blood-pressure. In such individuals it more often takes the form of a feeling of instability than of a sense of rotation, and is often intensified or precipitated by stooping. Circulatory disturbances are probably also responsible for the vertigo which may occur during an attack of migraine, and for the occurrence of this symptom in individuals with vasomotor instability at the onset of a syncopal attack, and as a reflex consequence of gastro-intestinal disorders. Treatment in such cases is that of the causal condition. Acute vascular lesions—for example, arterial thrombosis—may cause severe vertigo when the vestibular nucleus or cerebellum is involved. The classical example of this is thrombosis of the posterior inferior cerebellar artery.

(v) *Epilepsy*.—Vertigo may constitute the aura of an epileptic attack. Difficulty in diagnosis may arise when vertigo occurs as

the aura of an attack of *petit mal*, since giddiness may be the only symptom of which the patient is aware. The following points are of value in diagnosing *petit mal* from aural vertigo. The onset of an attack of *petit mal* is sudden. There is always some impairment of consciousness. The attack itself is brief, lasting only a few seconds, and recovery is immediate, there being no vertigo after recovery of consciousness. In aural vertigo, on the other hand, the onset of the attack is sometimes sudden, but is often more gradual than that of an attack of *petit mal*. Impairment of consciousness is very rare, and occurs only in the most severe attacks. The attacks are usually more prolonged, and recovery from the attack is more gradual.

(vi) *Neurosis*.—Vertigo is a common neurotic symptom. In neurotic patients giddiness usually takes the form of a sense of instability which is frequently persistent, though it may be described as occurring paroxysmally in an "anxiety attack," when it is associated with intense feelings of anxiety, sweating, tachycardia, and often tremor. Neurotic giddiness, however, is not in itself as a rule an incapacitating symptom, and its nature is recognised from its association with other neurotic symptoms and from the absence of other causes of vertigo. Psychological analysis usually reveals that the giddiness of the neurotic patient symbolises a fear of falling. It is a common symptom in patients who fear that they have heart disease which may lead to their falling dead in the street, but it may also symbolise a fear of financial ruin or a sense of mental or moral instability. Neurotic vertigo requires psychological investigation and treatment.

CHAPTER XV.

MINOR SURGERY OF THE EYE

BY

N. BISHOP HARMAN, M.A., M.B., F.R.C.S.

Consulting Ophthalmic Surgeon, West London Hospital.

OPHTHALMIC surgery is the most delicate of all surgery. The margin between success and disaster is the narrowest. It is therefore small wonder that for the most part its practice is in the hands of those who are ophthalmic surgeons first or solely. Nevertheless, there are some operations that any practitioner may be called upon to perform, and at a moment's notice. It is with these that I propose to deal. These minor operations done well will ensure the comfort and safety of the patient's sight and redound to the credit of the practitioner. Bungled, they may present serious risks to the patient, and leave a long memory of failure against the practitioner.

There will be considered certain features of the treatment of the conjunctiva: the methods of removal of foreign bodies; operations for chalazion; for some forms of corneal ulcers; and the examination of the lacrimal apparatus and the treatment of lacrimal abscess.

In all these states it is necessary to make a complete examination of the nooks and crannies of the conjunctiva, and facility in everting the lids is therefore needed. I may be forgiven for making some note on the manner of achieving this manoeuvre. The lower lid is easily pulled down, and when the patient looks up the conjunctiva covering lid and fornix is exposed. The upper lid, however, is not so easily dealt with. Yet it is the more important to examine, both by reason of the extent of the membrane lining it and the depth of the upper fornix, and its greater liability to disease and to harbouring foreign bodies. Instruments should not be used for the purpose. They frighten children. The eye-lashes should not be used to pull over the lid—young ladies resent the loss of these adornments; neither of these aids are necessary. To the practised

hand everting the upper lid is a simple and nice performance. It is scarcely more difficult than depressing the lower lid, and it can be done with as little discomfort to the patient. All that is needed is the worker's thumb and index finger, and *the patient's lower lid*. The manœuvre may be described thus: Stand facing the patient; use the right hand for the left eye, and the left hand for the right eye. Tell the patient to look down. Place the inner edge of the thumb parallel with the lower lid and on its outer half, just below the lashes. Place the inner edge of the index finger lightly on the upper lid just above the lashes, and gently push up the skin; this will cause the edge of the lid to lift from the globe and pout a little. Now with the thumb in position, slip the lower lid under the upper lid, and the thumb, following the lower lid, will get under the upper lid, which then can be held firmly between thumb and finger and everted, even against a vigorous orbicular spasm. The secret of the trick is to make the *lower lid* do the work of raising the upper; it acts as a wedge to separate the upper lid from the globe, and allow the thumb to follow through. When the upper lid is everted the deep fold of the upper fornix has still to be explored. Foreign bodies may be tucked away in its depth. The everted lid may be lifted. Or the fold may be made to pout outwards by gently pressing upon the globe through both the everted upper lid and the lower lid. But this method should only be adopted with young healthy eyes, and not with old eyes whose vessels may be unduly fragile.

Irrigating and Painting the Conjunctiva.

In certain forms of conjunctivitis, *e.g.*, ophthalmia neonatorum, frequent irrigation of the conjunctiva is imperative. It is the most important part of the treatment. No instruments that deliver fluid under pressure may be used, such as squirts and large irrigators, and no instruments should be thrust between the lids. It is sufficient to evert the lids with the fingers. The safest irrigator is the "undine" (Fig. 9). It is one of the larger sizes of the drop-bottles used in the pathological laboratory. The spout should be quite short, and guarded by a piece of rubber tubing. Normal saline or boracic lotion are quite efficient fluids. It is not the contents of the lotion so much as the mechanical action of washing that is beneficial. After an irrigation it may be necessary to apply some form of antiseptic lotion: this should never be strong. A mild lotion

frequently applied is the better practice. If it be desired to apply some form of silver preparation, the surgeon should apply it himself, and never leave its use to the nurse. It is as much a piece of surgical procedure as using a scalpel. The lids should be everted, and both at the same time; then the edges of the tarsal folds of upper and lower lids will meet over the cornea and afford it some

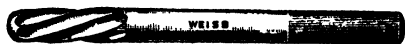


FIG. 1.—Glass probe for wool brush.

protection. Brushes should never be used for painting the lids. A fresh "brush" should be made for each application by the surgeon

by twisting a pledget of cotton-wool around the end of a thin glass rod or probe, and a piece of wax taper. The soft end of this cotton-wool brush can do no harm, none of it will be left behind, and we can be certain that it is sterile. If silver solutions are applied to the lids, no matter of what sort, it is well subsequently to wash out the conjunctiva with saline solution: this limits the duration of the action of the silver, and also removes fragments of epithelium that may be brought away from the membrane, and which if left under the lids cause pain.

Foreign Bodies.

A foreign body in the eye is an extremely common accident. The frequency of the happening in certain occupations makes the use of guards to machinery and of suitable goggles in dangerous trades imperative. Workmen are slow to learn that it is not cowardly to protect their eyes, whereas it is cowardly to run risks that may diminish their earnings and the living of their wives and children. The majority of foreign bodies are removed with ease—when once the fragment is found. The finding may be difficult. The youngest and best of eyes may not be able to see it without help. Binocular magnifiers are most useful for this purpose and for many operations about the eyes. They are only useful to those who have good binocular vision, *e.g.*, who can see the phenomenon of a stereoscope. These glasses give magnification both of surfaces and of depth. This is of particular value on the curved surface of the eye. The glasses are made up of +5D spheres cut in half: one half is placed before each eye, with the thin edge outwards and the thick edge towards the nose. The lenses must be worn as far from the eyes as possible, so a long nose is a good feature;

also the lenses must be tilted forward 15° , so as to be nearly in the place of the object to be viewed.

The inspection of the cornea is best performed by making use of the reflecting action surface of the cornea (Fig. 10). The patient should be so placed that the light of a window is mirrored on his cornea. Then he should be told to follow the movements of the surgeon's finger, so that the image of the window is seen upon every part of the cornea. If there be a foreign body on the surface, its presence and site will be seen without difficulty by the broken reflex—a wobble in an image that should be regular and stable. The removal

of the foreign body thus located is usually effected without difficulty with a spud after the instillation of cocaine. The choice of a spud is of some moment, for the foreign body must be removed with the least possible damage to the

corneal epithelium. If the accident be recent, my "niblick-shaped" spud may be used: the end is pressed on to the cornea beside the foreign body so as to depress the surface, then a forward movement will lift it off the cornea without scraping the epithelium. If the foreign body is embedded within the epithelium, a chisel-shaped spud should be dipped into the epithelium beneath the foreign body, and this, with some cells, should be lifted off. If the foreign body is fixed in the substance of the cornea, a needle must be used that will prick into the tissue beneath the foreign body and push it out; but this last manoeuvre should not be used except when it is imperative, for it damages the tissues, and may open up the corneal spaces to infection.

Every foreign body involves a certain element of risk of infection, and therefore irrigation should follow its removal. With older men there is not infrequently some sepsis of the lacrimal sac, and a foreign body in such a subject may be followed by hypopyon



FIG. 2.—"Niblick" spud.



FIG. 3.—Chisel spud.



FIG. 4.—Bowman's needle.

ulcer and serious damage to the sight. For this reason pressure should always be made over the lacrimal sac after the removal of a foreign body, and if there be the least suspicion of regurgitation, the sac should be syringed out, and the eye examined daily. If the sac be manifestly septic the puncta should be sealed up by the application of the actual cautery, and the sac laid open and drained, or else excised.

In rare cases a patient may be convinced that there is a foreign body in the eye, and yet none can be seen. Search should always be made for displaced eyelashes. Sometimes these get fixed into the lacrimal puncta and cause a local irritation. Minute wing-cases from insects may adhere to the cornea, producing a lesion like a phlyctenule.

Chemical foreign bodies, such as lime or acids, or the effects of exposure to flame, steam or hot water, produce intense reactions. In such cases the eye should never be washed with watery lotions, but some heavy neutral oil such as olive, cod-liver or castor oil, should be at once instilled. Pure cocaine dissolved in castor oil may be used to relieve pain. There must be continuous cold applications to the lids to reduce the intensity of the reaction. Care must be taken that the raw surfaces of the conjunctiva do not adhere. This is best effected by packing the lids with grease, and separating the lids daily with a glass rod.

Chalazion.

This small tumour is due to disorder of a Meibomian gland. These glands are set in the margin of the eyelids. They are the grease glands of the lids. They lubricate the margins, prevent tears running on to the face (except when in excess), and prevent soreness from blinking. The glands are long and tortuous. The chalazion is due to microbic infection. A granuloma is produced within the gland, and later this breaks down, producing a cyst-like gelatinous mass of cells. The chalazion is then said to be ripe. When in this state the appearance of the lump on the inside of the lid is characteristic. There is a dusky patch encircled with a rim of red. The dusky patch will be found, on touching it with a small probe, to be soft. Many attacks of this order will subside after the use of frequent hot bathing to the eye, or fomentations. Gentle massage may secure the discharge of the offending cells from the gland. But if these measures fail, then the mass must be evacuated by surgical means.

A few drops of cocaine and adrenalin should be instilled. After five minutes the operation may be done. The lid should be everted and held in a suitable forceps so as to prevent bleeding from the marginal artery. There is no more suitable forceps for this purpose than Graddy's expression forceps. They are better than those with complete rings and plates on the skin blade. Forceps with ratchets or clamps may be dangerous, since the lid may be gripped too severely.

The incision should be made with a fine, very sharp knife, and be vertical to the lid margin. It must never be parallel with the

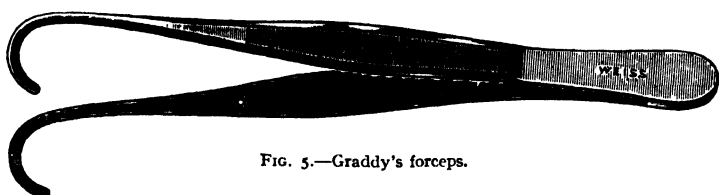


FIG. 5.—Graddy's forceps.



FIG. 6.—Spoon.

margin, for this will mean that other glands are cut. The next step is to scrape out the interior with a small sharp spoon. This is the painful part of the procedure, for the cocaine does not penetrate deeply. To diminish pain and make the scraping more effective, it is well to dip the spoon into some liquid pure carbolic. The spoon with its droplet of carbolic is then slipped between the lips of the wound deep into the mass of cells. It should be held there for a minute. The carbolic will effectively anaesthetise the interior. It can then be scraped out by quickly rotating the handle of the spoon between thumb and fingers. This is far better than attempting a direct scraping of the walls. The cell mass will be extruded, and may be wiped away. Care should be taken to see that none of the granulations is left between the lips of the wound. If the chalazion has been well scraped in this fashion there will be no subsequent bleeding. The conjunctival sac may be wiped clean with lint, the midriatic effect of the cocaine neutralised with a drop of eserine, and the patient may go home without a bandage. He should be told to bathe the eye frequently with hot water, and

to use some astringent drops, such as zinc sulphate. A chalazion is not a sty. A sty is a pustule on the outer edge of the lid margin in a sebaceous gland attached to a hair follicle. It can be emptied in many cases by pulling out the eyelash. Hot fomentation will promote evacuation. Occasionally it is necessary to pierce the centre with a small sharp knife. It is never necessary to scrape it out.

Lacrimal Troubles.

A tearful eye is a nuisance to the patient. He may wish for treatment. The first necessity is to discover the cause. The puncta may be inspected for position and shape. Pressure over the lacrimal sac will show if there be any excess fluid therein, for it will cause regurgitation. Since the duct drains into the nose, the interior of that cavity must be explored. Failing any evidence of obstruction at either end of the duct, the patency of the duct must be determined. The simplest way to determine this is to use a stain, and discover whether or no it passes from the conjunctiva into the nose. A fragment of dried fluorescein is best for this. It is placed in the lower fornix. If the tears are few these may be excited by nipping the columella of the nose. Then the pumping action of the eyelids should be exerted by the patient opening and then squeezing the lids together tightly; or the surgeon may distend the sac by pulling the lower lid away from the nose, and then by pressure over the sac force its contents down the duct. If the duct be patent the brilliant yellow dye will be seen when the nose is blown vigorously. The passage may take a minute or two, or in rare cases even as much as five minutes. If the dye comes through, the demonstration is dramatic. There is then no need for probing or syringing: astringent fluids are all that are needed.

If the dye will not come through, then it may be necessary to test the condition of the duct by other means. Syringing should be tried before probing. The punctum of the lower lid should be dilated so as to allow a fine syringe nozzle to enter it. The insertion of the nozzle first downwards and then inwards towards the nose will suffice for the first injection. If that fails it may be necessary to push the nozzle on until it is felt to stop in the sac at the wall of the nose. Again an attempt should be made to drive fluid through. It is far better to syringe out the sac for several days in succession before any attempt is made to probe the duct. Syringing

with hydrogen peroxide is very effective in recovering the health of a sac which is swollen and inflamed. Probing is of little use other than as a means of investigating the state of the duct. If this be deemed necessary it is better that it should be done by someone who is practised in the performance. A false passage may easily be made. In any case one should never syringe immediately after probing, for if a false passage has been made it is certain that the syringe will drive the fluid and possibly the contents of a septic sac into the tissues.

If there be an acutely inflamed lacrimal sac there is a clear indication for its opening. Treat it just as you would an acute abscess. Foment it freely. So soon as there is the slightest indication of pointing, or even before that happens if there be much pain, incise the sac. Pull the lower lid away from the nose and note the taut line between the inner canthus and the nose ; this is the line of the ligament that attaches the lids to the bone. Insert the point of the knife immediately below this line. If there be much pus, scrape it out with a spoon dipped in pure carbolic. If only blood comes out, slip a slice of the wall of a rubber tube into the cavity and foment freely. The operation causes acute pain, so needs a general anæsthetic.

Septic Corneal Abrasions and Ulcers.

After the removal of a foreign body, when there is a suspicion of sepsis or an actual ulcer, it is good practice to forestall the spread of the infiltration into the substance of the cornea by sterilising the site. The site of the damage must be clearly seen, and there is no better way to make it visible than by the use of fluorescein. After an instillation wait a few moments, then wash out the excess with saline solution. If there be an abrasion or ulcer it will be stained a brilliant green. To cauterise such an abrasion or ulcer there are no better fluids than pure carbolic acid, or equal parts of this with camphor. Cocaine may be used freely. But there is little actual pain in the operation, for the carbolic is in itself an efficient anæsthetic. The lids must be held apart by the thumb and finger of one hand, and one of the fingers of this hand should actually touch the globe so as to hold it steady (*see* Fig. 7, for position of the fingers). Next a pledget of cotton-wool should be pushed under the edge of the finger touching the globe, so as to absorb all the tears. The site of the abrasion may be dried by



FIG. 7 —Removing a foreign body—surgeon standing in front of patient. (Note position of fingers holding lids.)
From May and Worth's "Diseases of the Eye."

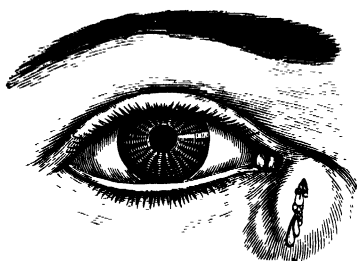


FIG. 8 —Lacrimal abscess opened

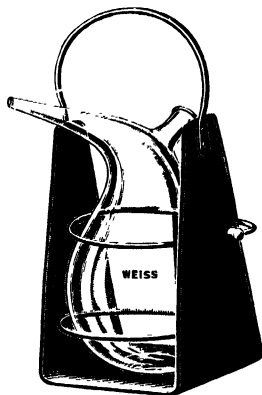


FIG. 9.—Undine in cradle



FIG. 10 —A window reflected in normal cornea ;
B—wobble in reflex due to foreign body ; C—foreign
body seen against fundus reflex.

"MINOR SURGERY OF THE EYE," by N. Bishop Harman, M.A.,
M.B., F.R.C.S.

touching it lightly with the flat surface of a piece of filter paper. The cauterising fluid is applied by means of a blunt-pointed piece of wood, such as a match. This is dipped into the fluid, and the soaked wood is stubbed into the ulcer surface. It is more necessary to stub the liquid into the margin of the ulcer than the surface. The parts touched turn white owing to the action of the carbolic. After the application again dry the site with filter paper. Then place some atropine ointment between the lids and bandage the eye. An hour later fomentation may be applied, and if the ulcer was of large surface and deep, it is well to apply leeches to the temple so as to reduce the reaction. For the most part this procedure should be used by the general practitioner as a preventive in cases where there is a suspicion of sepsis from a foreign body that he has removed. If there be a serious ulcer it had better be dealt with by one who is accustomed to such matters. The consequences of septic ulcers of the cornea are so serious that no risks should be taken with them.

I have described these operations as minor operations. But it may be taken as axiomatic that there is nothing little about them. They are as serious in their possibilities as an acute glaucoma. Each, if neglected, may destroy an eye. Again, if the practitioner has ambitions he may be assured that if he cannot treat these minor conditions with nicety he will never rise to the attainment of security in the treatment of more difficult conditions. Practice in the removal of foreign bodies and operations upon the lids is the best of training for the successful manipulation of eye instruments.

CHAPTER XVI.

THE TREATMENT OF PELVIC FLOOR PROLAPSE

BY

THOS. G. STEVENS, M.D., F.R.C.S., F.C.O.G.

Obstetric Surgeon, St. Mary's Hospital, London ; Surgeon, the Hospital for Women, Soho Square, London.

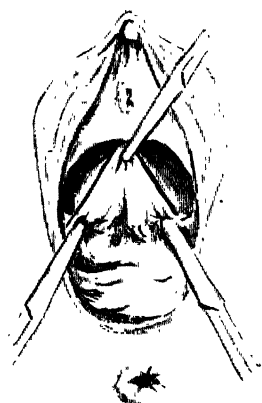
FORMERLY the term "prolapse of the uterus" was employed to include all the conditions which we now recognise as parts of a much wider lesion, which may be called "pelvic floor prolapse." Cystocele, rectocele, elongation, and hypertrophy of the cervix uteri, and descent of the uterus itself, all represent phases of pelvic floor prolapse ; but descent of the uterus does not necessarily occur at all, and in quite a large number of cases the uterus is more or less fixed at its normal position. When this is the case, cystocele, rectocele, or both combined, may be the only obvious features of prolapse, and consequently it is a misnomer to speak of all these cases as "prolapse of the uterus." Sometimes the uterus is the only prolapsed structure, moving up and down the vagina like the parts of a telescope, without any cystocele or rectocele. This variety is the least common of all the cases met with. A careful scrutiny of prolapse in general shows that it is characterised by descent of all the movable structures which make up the female pelvic floor, including the bladder, urethra, anterior vaginal wall, uterus, and finally the posterior vaginal wall. The fixed structures in the pelvic floor, namely, the levatores ani muscles, the rectum and perineal body, remain in place, or are merely stretched. Thus it appears that a rectocele, bulging of the anterior wall of the rectum over the internal sphincter ani, and carrying with it the posterior vaginal wall, is not a condition of descent at all, but is one of stretching and pouching of the rectum, and has a special causation of its own. Further, it may exist by itself without any

other manifestation of pelvic floor descent. It is the result of laceration of the perineum and healing without suture by granulation, thus fixing the posterior vaginal wall to the rectum by dense scar tissue. The rectum and vagina are then unable to slide over one another, so that during defæcation the mass of fæces always bulges the anterior rectal wall over the sphincter, and finally produces a definite pouch, which constitutes a rectocele. Cystocele, on the other hand, though associated with perineal tears, is really the result of wide separation of the two halves of the levator ani muscle and damage to the pubo-cervical muscle sheet, whereby the base of the bladder is deprived of its normal support, and so bulges downwards. Descent of the uterus depends upon two essential factors, stretching and laxity of its lateral supports, the transverse cervical ligaments (base of the broad ligaments and upper vaginal lateral attachments), which leaves the organ loose, and widening of the aperture between the halves of the levator ani muscle, which opens out the road down which it will travel. All these factors are the result of childbirth, and except in the very rare cases of prolapse in virgins, childbirth is the only cause. Elongation of the supra-vaginal cervix, which may play a great part in prolapse, is the result of the drag exercised by the vaginal walls, with cystocele or rectocele, when the uterus is more or less fixed. This fixation occurs when the transverse cervical ligaments are normal, or when one or both are inelastic from the scarring of an old pelvic cellulitis.

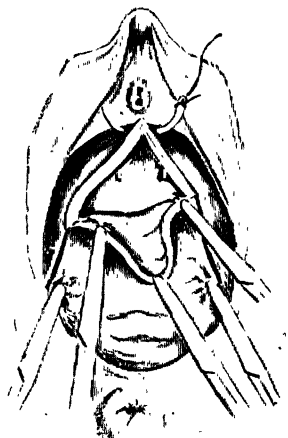
With so many and varied manifestations, it is obvious that all cases of prolapse cannot be treated in the same way. It must be recognised that if left alone all cases of prolapse inevitably become worse as time goes on: they do not improve at the change of life or by any palliative treatment. The only cure is by means of properly planned operations on the pelvic floor, according to the manifestations present. Many patients with moderate degrees of prolapse can be made comfortable with ring pessaries; but it cannot be denied that pessary treatment is not curative, that the prolapse becomes worse in spite of the pessary, and that this treatment has drawbacks of its own of an unpleasant nature. Offensive discharges, ulceration of the vaginal walls, and erosion of the cervix, to say nothing of the annoyance of daily douching, are amongst the troubles the pessary patient has to put up with. Other cases cannot be treated with ring pessaries because the vagina is funnel-shaped, with the wide end downwards, and no pessary will remain

in position. Thus it has come about to-day that more and more patients are treated by operative means. Formerly, a perfunctory perineal repair, in order to enable the patient to retain a pessary, was the only operation done. To-day operations must be curative, not only in an anatomical sense, but also as regards the patient's symptoms, the chief of which are backache, dragging in the groins, and various micturition troubles. An operation for prolapse which does not cure symptoms is a failure, however perfect the result may appear anatomically. It seems hardly necessary to reiterate that prolapse can be cured by operative means, and yet patients are not infrequently seen who have been told by doctors that operations are useless in their cases. Literally it can be said with truth that there is no case of prolapse so severe that it cannot be cured by properly planned operations.

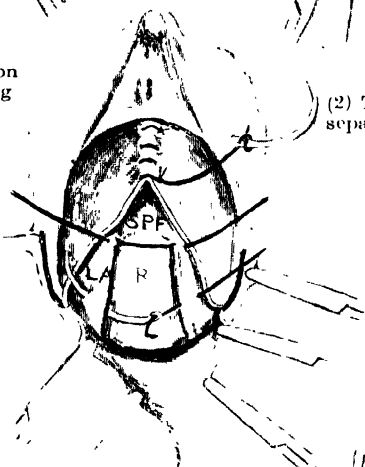
In Great Britain the modern operations for the cure of prolapse emanated from the Manchester school, and the names of Archibald Donald and Fothergill will always be remembered as the pioneers in this work. Before their time, operations naturally had been done, but they were rarely sufficiently thorough to be called curative, and more often than not consisted only of repair of a torn perineum. This did not always cure a rectocele, and cystocele was not remedied by it. Anterior colporrhaphy was known, but as it usually only ment the excision of an oval area of vaginal mucosa, with subsequent suture of the cut edges, it rarely succeeded in curing the lesion. To-day operations based on those invented by Donald and Fothergill are performed all over the world; certain modifications from the original operations have been introduced by individual operators, not always with any great advantage. One very great point must be insisted on, and that is *that it is very rarely necessary to open the abdomen to cure prolapse, and that with very few exceptions vaginal plastic surgery is all that is required.* Ventrofixation and operations like it are only necessary when a persistent retroversion is present, after vaginal plastic operations have been done; usually the latter will cure ordinary cases of retroversion. *Ventrofixation alone will not cure any case of prolapse,* as this operation does nothing to cure or prevent cystocele or rectocele. Thus it must be an axiom that vaginal plastic surgery is the first essential. The operations to be done comprise colpoperineorrhaphy, anterior colporrhaphy, and amputation of the cervix, where rectocele, cystocele and elongation of the cervix are all present. Rectocele alone usually requires



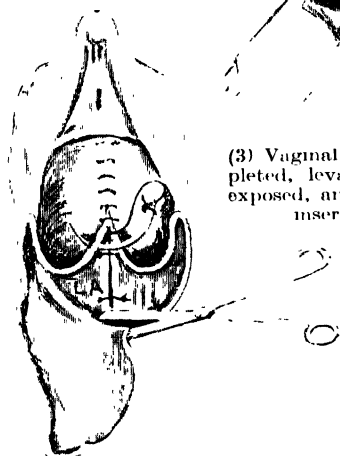
(1) Shows the incision at the apex of the vaginal flap.



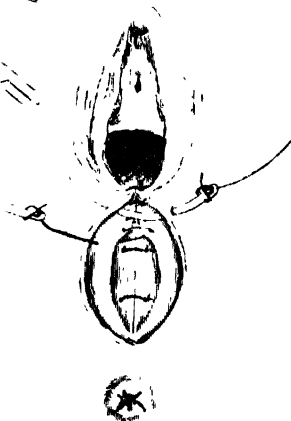
(2) The vaginal flap being separated from above downwards.



(3) Vaginal flap completed, levatores ani exposed, and sutures inserted.



(4) Sutures nearly completed, vaginal flap being cut away.



(5) Completion of superficial sutures. R, rectum; S.P.F., subperitoneal fat; L.A., levator ani. (T. G. S.)

PLATE I.—Colpoperineorrhaphy.

(By kind permission of the University of London Press, Ltd.)

"THE TREATMENT OF PELVIC FLOOR PROLAPSE," by THOS. G. STEVENS, M.D., F.R.C.S., F.C.O.G.

only colpoperineorrhaphy. These operations should be done at one sitting, when they are all necessary, but it is desirable that they should be performed fairly rapidly. There is a considerable amount of blood lost, and the longer the operator takes, the more blood is lost. Spurting vessels can be tied, but in general the hæmostasis is brought about by suturing, and therefore the more quickly this is done the better.

Colpoperineorrhaphy is best done by Donald's method, commencing high enough up the posterior vaginal wall to be above the rectocele. A forceps is clipped on at this point, which marks the apex of a triangle of vaginal mucosa to be removed, the base of which will eventually form the sides of the new perineum. Two other forceps are placed lower down and further out towards the sides of the vagina to mark the commencement of this triangle. Before making incisions just outside these forceps a catgut suture is tied in the mucosa just above the first forceps and left long. This prevents the mucosa going up out of sight when the triangle of mucosa is dissected downwards. The sides of this triangle are then united with a continuous suture, using the catgut already tied in. Forceps are again clipped on lower down and further out, and the triangle is stripped off the rectum from above downwards. Finally, forceps are clipped on the labia at a point on either side which will be the anterior border of the new perineum when suturing is complete, and the triangular flap now completely separated from the rectum is cut away with scissors. Two catgut sutures are placed deeply through the levatores ani muscles, preferably with a Reverdin's needle, and tied. Colles' fascia and its underlying tissue is then united with a continuous suture, and finally the skin is united in a similar manner. Thus all deep parts are united by buried sutures of catgut, and any dead spaces in which blood clot might collect are sutured here and there as may be necessary. These vary in each case, but are always obvious.

Commencing the operation from above is much easier than by the older method from below, and also has the advantage of starting above the rectocele. Starting from below, the operation is often so difficult that the upper part of the rectocele may never be reached.

Anterior Colporrhaphy.—Personally, I do not follow Fothergill's technique, but perform an operation which has the same result in the end. A straight incision is made between two forceps

clipped on the anterior vaginal wall, one near the cervix in the anterior fornix, and the other about three-quarters of an inch below the urethra. Two flaps are then dissected up, partly with the knife at first, and completed by rubbing the vaginal wall off the bladder with the finger covered with gauze. As long as this manoeuvre is carried out in the loose areolar tissue between the bladder and vagina, it is quite easily and quickly completed. The bladder is then picked up with dissecting forceps, and is completely separated from the uterus with scissors until it is quite free as far as the utero-vesical pouch of peritoneum. The bladder can then be pushed up out of the way. Suturing is now carried out by three mattress stitches of catgut placed in such a way as to bring the lateral vaginal walls to the centre; the furthest out suture is the one near the vaginal fornix, and when tied, has the effect of pushing the cervix back towards the sacral hollow. This suture also includes part of the base of the broad ligament, and all of them include the pubo-cervical muscle sheet. Thus these sutures bring a wide area of vagina together, and form a shelf on which the bladder will afterwards rest. Finally, redundant vaginal wall is cut away beyond the mattress sutures, and the cut edges are united by a continuous stitch. It will be noted that buried sutures are not used here—they are unnecessary, as the mattress sutures bring together all the deep tissues required.

Amputation of the Cervix.—When this is required on account of supra-vaginal elongation, the operation can be combined with anterior colporrhaphy. The straight incision up to the anterior fornix is carried round the cervix, and the vagina is dissected off the cervix as high as it is desired to amputate. The cuff of vagina is then sutured to the cut cervical canal either by a number of interrupted sutures, or by Sturmdorff's suture. The result in either case is the formation of the new external os. Hæmostasis is very important, and is brought about by tying vessels or deep suturing.

When completed the vagina is packed fairly tightly with a single strip of gauze introduced through a Kelly's rectal speculum. Damage to the suture lines is thus avoided, and the packing prevents oozing of blood from the suture lines. A firm pad is then placed on the vulva, and fixed with some pressure by a T-bandage.

As there is usually much pain for the first few days, morphia is used freely at night, and aspirin, in 10-grain doses, three times a day. Douches are not used, but efforts to keep the vagina non-

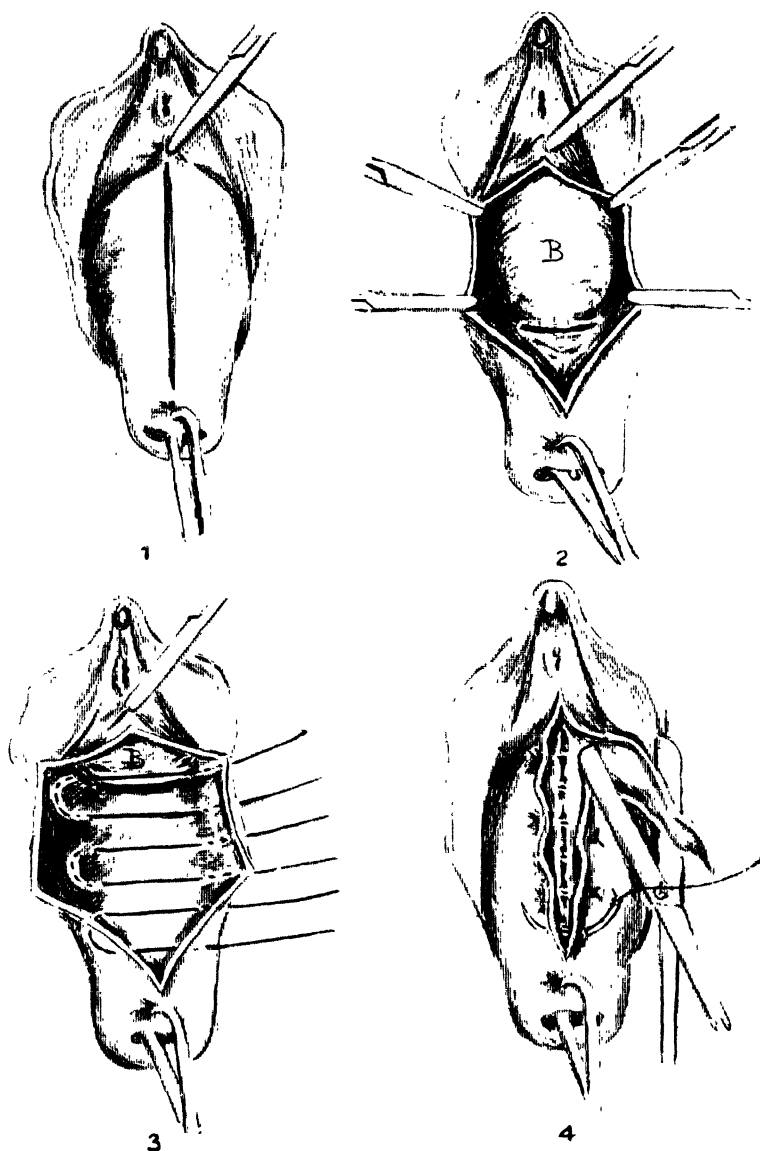


PLATE 2.—Anterior colporrhaphy. (1) Shows the incision in the anterior vaginal wall; (2) bladder (B) exposed, vaginal mucosa completely separated from it; (3) mattress sutures bringing together the paracervical and paravaginal tissues; (4) redundant vaginal mucosa cut away and superficial suture. (T.G.S.)

(By kind permission of the University of London Press, Ltd.)

"THE TREATMENT OF PELVIC FLOOR PROLAPSE," by THOS. G. STEVENS,
M.D., F.R.C.S., F.C.O.G.

septic are made by introducing each day two drams of 1 in 1,000 acriflavine solution, or a pessary of collosol argentum. The bowels are not moved until three days have elapsed, and then by castor oil ; enemata are not used. The patient is kept in bed sixteen days, and is instructed to sit in an arm-chair or lie on a sofa for another fortnight, avoiding the upright position as much as possible.

The only sequela of the operation of any importance is occasional secondary hæmorrhage, usually from a small artery in the posterior vaginal wall. This is stopped by packing the vagina, after clearing out clots through a Kelly's rectal speculum, with gauze soaked either in tinct. ferri perchloridi, two drams to one ounce of water, or with the first two inches soaked in one of the hæmoplastic sera. This hæmorrhage is caused by superficial sepsis and cutting out of the vaginal continuous suture. The same treatment usually suffices for secondary hæmorrhage from amputation of the cervix. Suturing under anæsthesia is rarely necessary for this complication. Deep suppuration is almost unknown, as there are no through-and-through sutures to convey sepsis to the deeper parts. Dyspareunia has been known to follow these operations if the vaginal entrance and passage are made too narrow. Naturally, care must be exercised to avoid cutting away too much of the vaginal mucosa.

CHAPTER XVII.

COCCAL INFECTIONS OF THE URINARY TRACT

BY

CLIFFORD MORSON, O.B.E., F.R.C.S.

Surgeon to St. Peter's Hospital for Urinary Diseases.

THE two organisms of this group which commonly invade the urinary tract are the staphylococcus aureus or albus, and various streptococci. In both urethral and prostatic obstruction of long standing these bacteria are frequently present in the urine, and are associated with chronic cystitis, which is very resistant to treatment. With regard to such cases, their presence is an indication of the permanency of the changes which have taken place in the bladder wall. Removal or relief of the obstruction will not cure the cystitis, though the infection can be kept under control by means of bladder lavage. Antiseptics such as potassium permanganate and flavine are most useful in this respect. These organisms are also constantly associated with secondary infections of carcinoma of the bladder and tuberculous cystitis. In both diseases the prognosis is seriously affected by this complication.

Recently the attention of urinary surgeons has been drawn to the condition known as carbuncle of the kidney. The disease is due to the staphylococcus aureus, which has invaded the renal cortex by way of the blood stream. The primary focus is a furuncle or carbuncle of the skin. The length of time previous to the formation of the kidney lesion is very variable. In some cases the patient has suffered from a crop of boils extending over a long period, and the onset of the renal complication follows immediately upon the resolution of the skin manifestation. Another case will give a history of boils on the back of the neck, long since healed. Rarely the patient has been treated for osteomyelitis, or a patch of eczema. It is stated that males are affected more

often than females, and certainly peripheral infections are more common in men than women. In some of the recorded cases there is a history of trauma on the affected side a few days before the onset of symptoms. It is extremely doubtful whether injury plays any part whatever in producing a renal complication of staphylococcal origin.

Pathologists recognise two types of staphylococcal suppuration of the kidney, one of which is termed pyæmia, the cortex being studded with small abscesses, while the other is the type called by Israel, carbuncle kidney. Whatever description may be given to this renal lesion, the cause is the same, and the treatment should be no different whether there are one or a dozen abscesses. The symptoms of this kidney disease have been clearly and accurately described by J. L. Joyce. They often begin suddenly with acute pain in the side of the upper abdomen and loin, which gradually gives way to a continuous boring ache in the subcostal region and back. In other cases the symptoms have a more insidious onset, with malaise and headache. The temperature is raised, and sometimes rigors occur. Flexion of the thigh is not unknown. There are never any urinary symptoms.

Physical examination reveals tenderness on deep palpation in the posterior renal angle on the affected side. Sometimes there is a little fulness of the abdomen confined to the area in the front of the diseased kidney. Movements of the lower part of the chest may be restricted, and auscultation will demonstrate diminished breath sounds at the base of the lung. If a perinephric abscess has formed, there may be œdema of the skin of the loin. The blood and urine should always be examined: the former for changes in the white cells, and the latter for the presence of pus cells and organisms. A polymorphonuclear leucocytosis is fairly constant, and cultures yield, as a rule, a pure growth of staphylococcus. An X-ray examination must never be omitted. In patients who give a recent history of some staphylococcal focus, such as a boil or carbuncle, the diagnosis is easy; but where there is no obvious peripheral lesion, considerable difficulty may be experienced. In the latter contingency, pyelography is extremely helpful, obliteration or blurring of the cup-shaped end of one or more calices being important evidence in conjunction with the physical signs. Such an investigation assists in excluding the possible presence of an inflamed appendix or gall-bladder. If the primary

focus can be discovered, treatment of it must precede that of the condition of the loin. Furunculosis should be dealt with on the usual lines, emphasis being laid on the necessity for opening the boil when it has formed pus, and never damaging the surrounding tissues by squeezing.

As regards the renal lesion, it is necessary to stress the importance of conservative treatment. The surgeon must be in no hurry to explore the perinephric tissues unless he is satisfied pus is present. In this respect help is afforded by needling the loin. Œdema of the skin is indicative of the attempt of the abscess to point through the muscles of this region. Should he decide on investigation of the condition of the kidney by operation, on no account ought he to perform nephrectomy. It is impossible to foretell that the other kidney will not become affected. Partial nephrectomy is unsound on account of the inability to determine the full extent of the lesion. It must be remembered that the patient is suffering from septicæmia, and therefore the main attack should be directed against the infection in the blood. The armamentarium for this purpose consists of good nursing, stimulants, and the intravenous injection of 20 per cent. sodium chloride. Brief reference must be made to the effect of septicæmia of streptococcal origin upon the kidneys. It is not sufficiently realised that in these serious cases the organism throughout the acute stage of the illness is being excreted in the urine. There are no urinary symptoms, and in the early stages very little change in the urine other than the presence of the streptococcus, a few leucocytes and albumen. Later, pus cells and casts are discovered, evidence of an infective nephritis.

When the patient has recovered from his illness, small calculi have been known to form in one or other kidney. These are without doubt of infective origin, and consist of calcium phosphate, and possibly dead bacteria. This is an added reason why the intake of fluids should be greater than normal throughout both the acute and convalescent stages of septicæmia. A forced diuresis will assist in controlling the infection of the kidneys, and if minute stones form, cause their dispersal without surgical aid and before they have reached any size.

CHAPTER XVIII.

THE TREATMENT
OF OBSTRUCTIVE JAUNDICE

BY

CHARLES NEWMAN, M.D., F.R.C.P.

Assistant Physician, King's College Hospital, London ; Assistant Registrar,
Royal College of Physicians.

JAUNDICE, as is always pointed out, is not a disease, but a symptom. This description is eminently valuable in that it stresses the importance of treating the cause of the jaundice rather than the jaundice itself. It has, however, the grave disadvantage of obscuring the possibility that the nature of the jaundice may alter after the diagnosis has been made. This is particularly true of the obstructive type. Obstructive jaundice is due to simple blocking of the bile ducts, the parenchyma of the liver being undamaged, in contradistinction to the toxic-infective variety, which is associated with damage to the liver cells themselves.

Now when the excretory duct in any gland of the body is blocked, the gland undergoes functional and, finally, anatomical destruction. The liver is no exception to this rule. Every clinician knows that obstructive jaundice is always liable to be complicated by cholæmia, which is fatal, although sometimes the jaundice may in itself be absolutely harmless. Cholæmia is the clinical aspect of liver-function failure, and failure of function is the ultimate fate of every gland whose excretory duct is blocked. The length of time required for the development of this complication varies enormously. In some cases, such as those in which the obstruction is incomplete, it may be very long ; in others, such as those in which the bile ducts are infected or the liver previously diseased, it may be very short. It is always difficult to estimate how long the obstruction may be left without danger. The biochemical tests, such as a failing sugar tolerance, the appearance of urobilin in the

urine, and a change in the Van den Bergh reaction, which may be helpful, are not available to most practitioners, and by the time the clinical evidences of cholæmia begin to appear, it is often too late to expect any treatment to save the patient's life.

What then are we to do? In the first place, I feel that it is very important that we should look upon obstruction to the common bile duct as a very serious state. For this reason operation, if it is ever going to be considered, should be considered at once, and the minimum possible time lost in watching a case to establish a diagnosis. Obstruction due to gall-stones should always be treated surgically if possible, as the stone responsible is usually of the facettèd type, which, being caused by inflammation, is very liable to set up cholangitis in the obstructed ducts, with rapid damage to the liver cells. If the obstruction is thought to be due to malignant disease, no matter where or of what kind, I would recommend a laparotomy. There are two reasons for this: First, that even the most confident diagnosis may be wrong, and there is nothing more distressing than to find, post-mortem, that such a patient had died from hydrohepatosis due to a simple stricture of the bile duct or a primary carcinoma the size of a pea, which could have been cured without difficulty by operation. Second, even if the obstruction is due to an inoperable malignant disease, the gall-bladder can be anastomosed to the intestine or drained, and the patient will not be left to die with all the added unpleasantness of obstructive jaundice. Even if the patient should fade quietly away in a few days as a result of the operation, I think most surgeons who prefer humanity to their statistics would consider they had done a good work.

In the next place we must apply treatment to prevent the failure of liver function. For this purpose we have now various means.

(1) *Glucose*.—The liver cannot work without an adequate supply of glucose, and it is extraordinary how efficient this substance is in treatment. It is best given as such, and the cheap commercial syrup is just as good as the very costly crystalline form. It may be given as lemonade or any other fruit-juice drink, the glucose being used as the sweetening agent. This is very palatable to a jaundiced patient, and enables large quantities to be given without causing nausea. Grape-fruit juice or pineapple juice makes a very pleasant change from ordinary lemonade, of which patients soon tire. If glucose is unobtainable, cane sugar is practically as

good. In cases of urgency (when cholæmia has set in, for instance) it is best to administer a 20 or 30 per cent. solution of glucose intravenously. One or two hundred c.c. may be given at a time, followed advantageously by saline (up to 500 c.c.) before withdrawing the needle. For intravenous use the solution must, of course, be sterilised, and cane sugar must on no account be used.

(2) *Alkalies*.—The liver is extremely easily damaged by either acidosis or ketosis, and it is advisable to give all patients with obstructive jaundice 10 to 15 grains of sodium bicarbonate three times a day. It is important not to give too much, since this may actually lead to ketosis. If the urine becomes alkaline, reduce the dose. There is no need for it ever to pass neutrality.

(3) *Calcium*.—It was found that calcium exerted a protective effect on the liver in experimental animals, and although its efficacy has not yet been proved in human beings, its results are sometimes extremely gratifying. It may be given by mouth in the form of calcium sodium lactate (Kalzana), but is much best given intravenously. For this purpose calcium chloride is to be avoided, except by the very experienced; the calcium sugar compounds are very much to be preferred. Calcium levulinate may be obtained in ampoules containing 2 to 5 c.c. of solution ready made. Give 2 c.c. the first day, and 5 c.c. on the second, fourth and sixth days, and after that every third day. It is best to use an ordinary hypodermic needle for the intravenous injection, and it should be given very slowly, taking three minutes over the 2 c.c. injection and at least five minutes over the larger one. It should not affect the heart theoretically, but I have seen the pulse slowed alarmingly on one occasion by an injudiciously rapid injection.

(4) *Diet*.—It is scarcely necessary to say that the diet should consist of fluids, starches and sugars, and that meat and fat should be avoided; but it is perhaps worth mentioning that soups and other meat extractions, so dear to the hearts of relatives, are better interdicted. Fruit of any sort and quantity may take their place. In any case it is wise to remember that the patient's own appetite is a better guide than a doctor's theories.

Treatment of Pruritus.

The administration of thyroid extract is often advocated, and is certainly harmless, but it has not, in my hands, been very satisfactory. I have seen far more benefit from soaking the patient

for twenty minutes in a comfortably warm bath (kept warm by periodical additions of hot water) to which has been added a pound of bicarbonate of soda or two ounces of washing soda. The former is better but expensive. This soaks the irritant out of the skin, and often leads to a night of sleep instead of a night of scratching. Antipruritics are practically useless, as it is impossible to apply a strong enough lotion to the whole body surface.

Treatment of the Digestive Disturbance.

In addition to the diet and sodium bicarbonate, patient are much comforted by half a minim of peppermint oil and an ounce of infusion of rhubarb. This may be given, with the bicarbonate, three times a day before meals. The best aperient is a saline in concentrated solution. This helps to eliminate poisons by the bowel. It is useless to give calomel because of its supposed (but non-existent) cholagogue action, and I would similarly deprecate the use of salicylates and hexamine, which do the patient no good, and make the doctor feel that he is doing more than he really is, usually to the neglect of more useful measures.

CHAPTER XIX.

THE TREATMENT OF PLEURISY

BY

J. F. HALLS DALLY, M.A., M.D., M.R.C.P.

Senior Physician to the Mount Vernon Hospital and to the
St. Marylebone General Dispensary.

PLEURISY is not infrequently met with in the course of a large general practice, and although many cases are easily dealt with, others are liable to cause trouble and anxiety. Notwithstanding that evidences of past disease of the pleuræ are discovered in almost 75 per cent. of all autopsies, yet clinically manifestations of this malady are often absent, or so slight as to remain latent and undiagnosed.

The vast majority of cases of pleural inflammation are undoubtedly infective in origin; seldom primary, apart from tuberculosis, but generally secondary to some previous focal infection of the lung (tuberculosis, pneumonia, bronchitis, abscess, gangrene, etc.). Certain cases arise (*a*) as a result of direct extension of disease from adjacent organs other than the lung, or from serous membranes (pericarditis, peritonitis); (*b*) *via* the blood stream, as a part of a general infection (septicæmia, pyæmia); (*c*) in the course of infections of undetermined origin (rheumatic fever, scarlet fever); (*d*) by the agency either of organisms or of chemical toxins in chronic disorders of metabolism (gout, diabetes, chronic nephritis); (*e*) by metastasis from more distant focal infections (tonsillitis, appendicitis, oral sepsis); (*f*) traumatic (wounds, contusions, fractures of ribs); (*g*) from infarction or new growth of the lung; (*h*) as complications (bronchiectasis, hydatids, etc.); (*i*) terminal, often in elderly subjects.

Bacterial Ætiology.—The most frequent causal agent is the tubercle bacillus. In 75 per cent. of cases of exudative pleurisy a tuberculin test will yield a positive reaction, while in 30 per cent. of such cases careful examination of the aspirated fluid will reveal

the presence of tubercle bacilli. If there be any sputum, repeated tests for tubercle bacilli should be instituted, and the chest examined at regular intervals during the next two years as a prophylactic precaution against tuberculosis.

Non-tuberculous exudative pleurisy, which occurs in about 18 per cent. of all cases, is most frequently due to the pneumococcus or streptococcus. Other organisms concerned are few, and can rarely be demonstrated in the exudate. A knowledge of the above data is of considerable value alike in diagnosis and prognosis. In practice, nevertheless, treatment depends less upon causal considerations than upon the course and degree of the individual manifestation. A ready clinical classification is discoverable in the nature of the effusion. Thus a dry pleurisy connotes a fibrinous exudate ; a pleurisy with simple effusion, one that is sero-fibrinous ; empyema, one that is definitely purulent.

Spontaneous Recovery.—The large proportion of cases of latent pleuritis unrecognised during life, or with slight symptoms—apt to be mistaken by the patient for minor ailments such as muscular rheumatism or indigestion because of pain referred to the region of the shoulder—teaches that this malady undergoes frequent natural arrest or cure. Many cases are probably of pure tuberculous origin, and therefore mild, changes in the pleuræ resulting therefrom being similar to those of tuberculosis in other tissues. Thus, in process of healing, nodules become encysted by dense fibrous tissue, into which granulations are also converted, with possible obliteration of the pleural sac and complete arrest of the disease.

Treatment.

(1) *Acute Dry Pleurisy.*—In the early stages treatment is necessarily symptomatic and akin to that of other inflammatory conditions. The usual symptoms are pain in the chest, aggravated by coughing or attempts at deep breathing, with slight or moderate fever. The characteristic physical sign is a fine friction sound, audible over the area affected.

General Management.—To promote early resolution, complete rest in bed from the onset, in a room that is comfortably warmed and to which plenty of fresh air is allowed access, is essential. Diet should be graded from a milk diet to one that is moderately full, in accordance with the state of the patient's tongue, stomach, and bowels. The latter should receive attention, a dose of calomel,

if tolerated, being administered at the beginning, followed by salts in the morning. The inflamed pleural surfaces should be afforded rest to the greatest extent possible by muscular relaxation, avoidance of deep breathing, and reduction of coughing and talking to a minimum.

Asthenic patients, with bounding pulse, if seen in the earliest stage of the malady, will benefit by small doses of tincture of aconite or veratrum viride, min. 2, every quarter of an hour during one hour, afterwards repeating the dose hourly until the skin begins to act freely and the temperature abates. Such remedies should, however, only be employed in the case of robust patients with full pulses, and should be discontinued as soon as the circulation becomes relieved. For those with feeble circulations diffusible stimulants are indicated.

Pain. (a) *Local Applications.*—Mild cases are relieved by thermogene wool, used dry, or rendered more potent in action by sprinkling with warm salt and water. More severe cases find alleviation by the employment of the official cataplasma kaolin (B.P. 1932), which, when spread on lint to the thickness of half an inch, is soothing and efficacious. Failing this, turpentine or belladonna stupes, or hot linseed meal poultices, find some advocates. Strapping the chest, which in former days was a usual routine, except for cases in which pain is intense, is less to be recommended, since it tends to embarrass the sound side, prevents effective auscultation, and in removal is apt to cause pain and disturbance to the patient. Dry cupping and leeches, good old-fashioned remedies, have again come into favour. In applying leeches, however, there are a few points which should be known and observed. They are usually sent from the chemist in a thin wooden box, but if sent in water, should be removed from this about half-an-hour before they are to be used. The skin of the patient is previously washed with water, but no soap, and the selected spots covered with a drop of milk, or scratched so that a drop of blood exudes. The leech should be applied by means of a leech glass, which consists in a short glass tube narrowing to one end, which is slightly curved. Failing this, a test-tube, or inverted wineglass, may be utilised. Since each leech draws a teaspoonful or so of blood, two are usually sufficient, and are allowed to suck for about half-an-hour. When filled, they drop off, or can be dislodged by sprinkling with a little salt. The presence of hirudin (leech extract) in the tiny rectangular

bite prevents clotting, so that continuous trickling of blood takes place subsequently, and the doctor should not leave the case until he is satisfied that bleeding has been checked by a styptic, such as powdered alum, applied in combination with firm pressure. For very intense pain, a good procedure is to introduce into the pleural sac a small quantity, say 200 c.c., of filtered air, which, by separating the inflamed pleural layers, acts as a cushion, and gives instant and complete relief.

(b) *Drugs*.—At the beginning, for slight pain, Dover's powder, 10 grains, is excellent. Aspirin, 10 grains, with or without pyramidon, 5 grains, may be given. Internally, an alkaline diaphoretic and diuretic is of use, such as the following: pot. cit., gr. 20; liq. ammon. acet., drams 2; syrupi limonis, dram 1; aquam camphoræ, ad oz. $\frac{1}{2}$, six-hourly. If the pain increases and the patient becomes restless and irritable, it may be necessary to have recourse to veramon, 6 grains, repeated if necessary by mouth, or to a hypodermic injection of morphia, $\frac{1}{8}$ to $\frac{1}{4}$ grain.

Insomnia.—Treatment of this symptom will necessarily vary as to whether or not it depends on a physical cause, such as pain, dyspnoea, commencing effusion, or extension of tuberculous disease, or on a psychical cause. If no cause is obvious, in mild cases 15 grains each of bromide and chloral hydrate will suffice; in moderate cases, allonal one to two tablets, or medinal 5 to 10 grains; in worse cases, omnopon, $\frac{1}{2}$ grain orally or hypodermically.

Cough.—This is usually dry, persistent and ineffective, and requires the exhibition of a suitable linctus such as glycerini, min. 20; syrupi limonis, min. 15; aquam, ad dr. 1, to which may be added, if necessary, heroinæ hydrochlor., gr. 1/20, or liq. morphinæ hydrochlor., min. 3.

(2) *Dry Chronic Pleurisy*.—Here resolution may be hastened by counter irritation in the form of mustard plasters or flying blisters, or by painting the chest wall with equal parts of the liniment and tincture of iodine. Internally a mixture may be prescribed containing the following: potassii iodidi, gr. 10; sodii bicarb., gr. 20; sodii salicylatis, gr. 10; ammon. carb., gr. 3; aquam chloroformi, ad oz. $\frac{1}{2}$, ter die.

(3) *Acute Pleurisy with Simple Sero-Fibrinous Effusion*.—After a week or so, fever, cough and pain may subside, and a slight serous exudate become absorbed. The process may be quickened

by painting the side with iodine, or by dry cupping. If the exudate does not exceed moderate limits, treatment is similar to that for acute dry pleurisy. A light diet with limitation of total fluids is advisable, the bowels being kept open with concentrated salines. Effusions extending above the fourth rib anteriorly, if left alone, naturally tend to take longer to absorb than those of less amount, disappearance of the fluid being accompanied by crackling or creaking friction sounds. For many months at the base of the affected lung resonance is impaired, and breath sounds but feebly audible. If after three weeks the fluid level stands at the same height, or is increasing—particularly if above the fourth rib in front—or if displacement of organs causes circulatory or respiratory embarrassment, after determination of the nature of the fluid by an exploratory puncture, siphonage or aspiration should be undertaken, unless the fluid is hæmorrhagic.

Paracentesis Thoracis.—Following preliminary sterilisation of the patient's skin with alcohol, a 1 to 2 per cent. solution of novocain is injected hypodermically into the eighth interspace at the angle of the scapula, the patient's arm being brought over to the opposite shoulder. The needle should be introduced by steps, releasing about 0.25 c.c. of the solution below the skin surface, and similar amounts successively at deeper levels downwards and inwards towards the base of the lung.

Siphonage.—In the presence of thin fluid, aspiration is not necessary, for the fluid will flow readily through a stout needle connected with three feet of rubber tubing, the whole being previously sterilised, filled with sterile water, and clamped. As soon as the needle has been inserted, the clip is removed, whereupon the effusion flows into a receptacle placed on the floor by the bedside.

Aspiration.—For thicker exudates an aspirating bottle becomes essential, so as to create a slight negative pressure. In order to obviate the liability of the pleural sac filling up again with fluid, which sometimes happens, a useful method is, simultaneously with withdrawal of the fluid, to introduce into the second interspace anteriorly a fine needle leading to a glass tube containing sterile cotton-wool, which serves the purpose of an air filter. By this means the escaping fluid becomes replaced by air, the lung at first remaining collapsed, but later undergoing gradual expansion relatively to absorption of air that has been introduced. As a

further precaution against return of the effusion, as soon as the intrapleural pressure has become equal to the atmospheric pressure the air filter may be replaced by a small funnel, and 3 c.c. of 1 in 1,000 adrenaline chloride solution, diluted to three times the amount with normal saline solution, allowed to flow through the tube and needle into the pleural sac.

CHAPTER XX.

PLEURAL EFFUSIONS—SEROUS AND PURULENT

BY

A. J. SCOTT-PINCHIN, M.D., F.R.C.P.

Physician, City of London Hospital for Diseases of Heart and Lungs.

MANY and diverse papers and opinions appear on this subject, often expressing quite contrary views, and one may therefore reasonably assume that the practitioner may be in some doubt as to the procedure he should adopt in any special case. To those whose contact with a hospital devoted to diseases of the chest brings them into constant association with these conditions, coupled with the fact that there is constant interchange of ideas with one's colleagues, the procedure becomes so much a matter of routine that it is apt to be forgotten that there are many points which may be misunderstood or difficult to the practitioner whose field is so much more wide.

I shall endeavour, without going into niceties, to put before my readers certain points which may be of use, and some which are of importance. Firstly, a few words with regard to tapping or exploring a chest. It is well never to needle a chest lightheartedly; fortunately complications seldom occur, but when they do they are alarming, and may be catastrophic. Personally, I never explore a chest without some trepidation and an uncomfortable sense that the unexpected may happen. Pleural shock is a bogey seldom met with, and yet on two occasions I have known patients with malignant disease and effusion suddenly die as a result of this procedure. Tapping too low on the left side may damage the spleen, causing bad hæmorrhage; I have seen this happen more than once. Needling a solid lung may cause catastrophe. I know of two cases in which the needle, passing through blood vessel and bronchus, produced fatal hæmorrhage. Mostly these

are complications one cannot foresee, or, except in the case of the spleen, avoid.

In exploring the chest a local anæsthetic should always be used ; it is probably a safeguard against shock, though it was used in both the above cases. Nevertheless, it is a precaution for the safety of the patient which should always be taken. The anæsthetic solution may be novocaine, percaine, phenolaine, or others. Patients rarely are susceptible to novocaine, and are very seedy with temperature after its use ; I have known this in several patients having pneumothorax refills. It is difficult to obtain real surgical cleanliness of the hands in an ordinary house, so that after washing it is better to dry the hands ; with dry hands there is little danger of infection if the business parts of the instruments are not touched. The skin is iodined, and a subcuticular bleb of the solution introduced to serve as a land-mark—this is all the patient should feel ; the needle is gradually advanced, injecting the fluid the while until the pleura is reached. If fluid is present, it can usually be felt by the absence of resistance, and if the syringe has been emptied of anæsthetic on its way in, a small amount of fluid, if present, may be withdrawn into it for diagnostic examination.

Slack methods of withdrawing quantities of fluid with a 20 c.c. syringe should not be indulged in ; some air is bound to enter each time the syringe is removed, and this air may be a nuisance or even a danger later. If it is necessary to draw off fluid, some form of aspirator should be used—Potain, Dieulafoy, or the simple two-way ball-valve junction such as is used on a suction pump. A trocar and cannula is preferable to a needle, with which latter there is a chance of wounding the lung as the fluid is withdrawn. The advantage of the Dieulafoy is that if an air replacement is to be done, air can be injected in known quantity through the syringe ; the disadvantage is that if the tap is not turned each time, the fluid or air may be pushed back into the chest. If for any reason it is necessary to remove all the fluid or much of it, remember that cough is a danger signal which may signify commencing oedema of the other lung, and stop the procedure instantly. Bear in mind that complications may be induced by tapping inadvisably. In abscess of the lung an empyema may be started, or, if air is introduced accidentally, may start later, jeopardising the chances of an already very sick patient.

Effusions into the pleural sac can be divided into : Transudates, inflammatory effusions (serous and hæmorrhagic), potentially purulent effusions, purulent effusions.

Transudates are fluids of low specific gravity, and are either due to systemic disease or pressure. The treatment is of the general condition, cardiac or renal as it may be, though sometimes it may be necessary to withdraw the fluid as part of the treatment.

Of the serous inflammatory effusions, there is no doubt the great majority are of tuberculous origin. Rheumatism would appear to be a cause, though this is not universally accepted. There are curious resemblances between the two diseases, tubercle and rheumatism : the same two types of patient are met with, they are both apt to be the subjects of erythema nodosum, and the diseases are seldom met with together. Those cases in which one has imagined the effusion to be rheumatic have taken very much longer to clear up than tubercular ones, and are usually associated with a chronic pneumonic condition in the lung. It has been suggested that a pleural effusion in tuberculosis is a sort of protective effort to splint the lung, and that this effect should be kept up as long as possible. There is no evidence to show that this is an effort of nature to produce immobilisation ; in fact pleural effusions are uncommon in parenchymal disease ; moreover, the lung is not usually found to be diseased in cases of pleural effusion at the time of the effusion, the disease in the lung often not appearing for many years. Probably at the time of the effusion the disease is glandular and pleural only, and there is nothing to be gained by keeping the lung compressed.

The fluid level in a case of effusion should be carefully watched, and if it increases so much as to cause distress in breathing, or much displacement of the heart and mediastinum, some should be drawn off. Cytological examination should be done from this specimen ; it is not necessary to take off much of the fluid—there is general agreement on this—except as regards air replacement, of which more later.

It is my practice in cases of tubercular effusion to have the fluid examined, and if T.B. are not present, a small quantity of the fluid—10 c.c.—is drawn off each week and injected into the subcutaneous tissues ; I believe the absorption and course of the disease is hastened by these means. I have never seen any harm come from this procedure, though French authors are against it.

This method of treatment would seem rational ; improvement is usually seen after drawing off a small quantity of fluid, after which there is probably a leakage of fluid along the needle track ; the same improvement is seen in tubercular ascites after opening.

Usually, after the withdrawal of a small quantity of fluid or auto-sero therapy, as stated above, absorption begins to take place and the patient may be left alone, but in some cases a second tap may be necessary. There are several points in favour of leaving the fluid : firstly, antibodies may be present in the fluid ; and, secondly, the mechanical effect. If a lung has been forcibly collapsed with a pressure of fluid, it does not expand freely at once if all the fluid is drawn off. Apart from the mechanical disturbance to the other lung, the chest wall fails to meet the lung, and retains this position after the lung has come out again ; therefore there is a permanent reduction in the volume of the thorax on that side.

There are those who suggest that all the fluid should be drawn out and replaced by air, on the assumption, firstly, that the fluid is toxic, and, secondly, that the lung should be kept collapsed. If the pleural exudate were secondary to pulmonary disease this would be justified on the analogy of artificial pneumothorax treatment, but in most cases the lung does not appear to be diseased at the time of the effusion, pulmonary disease not appearing in most cases till some years after the effusion, during which time the patient has usually had perfect health. The assumption that air replacement is advisable in all cases would seem to be based on a false analogy. Nevertheless, if there is a suspicion of pulmonary disease, either from X-ray, which often in these cases is misleading, or from the fact that T.B. are present in the sputum, air replacement is good treatment. A Dieulafoy aspirator can be used, and for two syringes-full of fluid one of air may be injected. After two lots of air have been injected, a pneumothorax needle connected to pressure gauge is entered at the upper part of the thoracic cavity ; if this is done before any air is introduced the fluid present may cause inconvenience, thereafter the pressure during the procedure may be watched, and should be left well on the negative side of atmospheric.

Hæmorrhagic effusions are met with in tuberculosis, influenza, neoplasm and other infected conditions of the pleura ; they are rare in tuberculosis, and probably the frequency of incidence is in the reverse order to which they are mentioned.

Purulent Effusions.—The pleura has strong antibacterial powers for the dealing with infection ; it is on this account that one so frequently finds that fluid which contains organisms resolves without going on to empyema. In the consideration of the treatment of purulent effusions, which are of themselves dangerous to life, the position is very different from that which obtains in the serous type. The present and future must be weighed in the balance ; it is to some degree a mechanical problem. In any pneumonic condition it is more frequent than not for there to be a certain amount of effusion : this is often non-infected, but in some cases becomes so later, forming an empyema. This point is important in view of recent suggestions that artificial pneumothorax is a good method of treatment in pneumonia. A pneumonic lung has no power of expansion, and during the absorption of exudate becomes smaller before it recovers its elasticity. If, therefore, a chest is opened with a syn-pneumonic empyema, an open pyo-pneumothorax is produced with a lung which has no expansile power and no aid to cause it to expand again—for we must remember that the closure of a draining empyema is effected by the expanding lung gradually filling the pyogenic space ; we should wait until there is some evidence of expansion of the lung tissue ; in the meanwhile the pus may be aspirated, or in some cases closed drainage may be attempted.

The same condition obtains in those cases in which a large effusion is rapidly formed without much local reaction, as in streptococcal empyemata with or without pneumonia. In these cases the lung is firmly collapsed without adhesions, and if this type of case is opened we are left with a cavity which shows little tendency to close ; it is either a question of months, with much permanent damage to lung expansion, or a severe plastic operation. It must be remembered that when the thorax is opened the pressure on both sides of the lung is nearly equal, and if the lung is collapsed the elasticity tends to contraction rather than expansion ; later exudation on the surface binds it still further, so that in chronic cases a decortication of the lung may be necessary. Sometimes in a case which has been opened at this unpropitious stage it is possible to put constant suction on to the open thorax by closing the opening with some adhesive, and so help the lung to expand by producing a negative pressure in the thorax. The saving grace in these cases is adhesions. If these are present they prevent

complete collapse, and give a means by which the lung can climb out again, as is well seen in artificial pneumothorax with adhesions or obliterative pleurisy, in which cases the lung comes out against pressure.

In cases of syn-pneumonic pneumococcal empyema and streptococcal empyema, aspiration should be continually employed until in the first case the pneumonic condition is subsiding and the lung expanding, or, in the second, the pus has thickened and adhesions have formed. Closed drainage by means of some suction apparatus, a tight catheter being left in the chest, is an alternative method. In the case of stinking empyema, where the infection is so severe that the toxic absorption is the most serious factor in the case, we are forced to drain and face the difficulties which may arise at a later date.

CHAPTER XXI.

SEASONAL HAY-FEVER AND ITS TREATMENT IN THE BRITISH ISLES

BY

GEORGE W. BRAY, M.B., Ch.M., M.R.C.P.

Assistant Physician, Princess Elizabeth of York Hospital for Children; Research Physician, Asthma Clinic, Guy's Hospital; late Asthma Research Scholar, the Hospital for Sick Children, Great Ormond Street, London.

SEASONAL hay-fever or pollen allergy refers to the train of symptoms affecting mostly the upper respiratory tract and eyes, though in many cases the lower respiratory tract later becomes involved, induced in specifically sensitive individuals on adequate contact with specific pollens. Its prevalence and intensity therefore depend to a large extent upon the botanical flora in the locality of the sufferer. The term "seasonal" is purposely included, as the present discussion leaves out of consideration those "perennial" cases who suffer from similar symptoms all the year round, due mainly to sensitisations other than pollens.

In the British Isles hay-fever is a very distressing and prevalent complaint, and no doubt this fact explains why a great deal of the pioneer work in its study originated in England. It was Bostock, first of Liverpool and later of London, who was the first (1819) to describe hay-fever as a "summer catarrh," a disease with a definite symptomatology and a seasonal occurrence. From 1859-1873, Blackley, of Manchester, by a series of most thorough and systematic experiments, proved conclusively that the pollen of grass was a causative agent. Noon and Freeman, of London, were the first to apply the routine conjunctival test in diagnosis, and in 1911 were the first to use pollen solutions for therapeutic purposes.

Hay-Fever-producing Plants.

Most of the plants that lead to hay-fever symptoms are widely and abundantly distributed, with small unscented flowers lacking

brilliant colourings. The pollen, the germ-cell of the male plant, is generally wind-borne rather than insect-borne, fairly prolific in its production, and sufficiently small and buoyant to be carried considerable distances.

In England the pollen cloud abounds from about the middle of May until the middle of July, becoming slightly later as one travels

	Feb.	Mch.	April	May	June	July	Aug.	Sept.
TREES.								
<i>Corylus Avellana</i> (Hazel)	xx	xxxx	xxx					
<i>Populus nigra</i> (Poplar)		xxxx						
<i>Fagus sylvatica</i> (Beech)		xxx	xxx					
<i>Ulmus campestris</i> (Elm)		xx	xxxx	xx				
<i>Betula verrucosa</i> (Birch)			xxxx	xx				
<i>Quercus Robur</i> (Oak)			xxxx	xx				
<i>Carpinus betulus</i> (Hornbeam)			xxxx	xx				
<i>Juglans regia</i> (Walnut)			xxxx	xx				
<i>Fraxinus excelsior</i> (Ash)			xxxx	xx				
<i>Plantanus acerifolia</i> (Plane)			xxxx	xxxx				
<i>Acer Pseudo-platanus</i> (Sycamore)				xxxx	xx			
<i>Acer campestre</i> (Maple)				xxx	xxx			
GRASSES.								
<i>Alopecurus pratensis</i> (Meadow foxtail)			xxx	xxx				
<i>Anthoxanthum odoratum</i> (Sweet-scented vernal)			x	xxxx	xx			
<i>Bromus mollis</i> (Soft brome)				xx	xx			
<i>Poa annua</i> (Annual meadow)				xx	xxxx	xxxx	xx	
<i>Poa pratensis</i> (Smooth-stalked meadow)				xx	xxxx	x		
<i>Poa trivialis</i> (Rough-stalked meadow)					xxx	xx		
<i>Dactylis glomerata</i> (Cock's foot or orchard)					xxxx	xxx		
<i>Lolium perenne</i> (Perennial rye)					xxx			
<i>Arrhenatherum avenaceum</i> (False oat)					xxx	xx		
<i>Holcus lanatus</i> (Velvet or Yorkshire fog)					xxx	xx		
<i>Festuca ovina</i> (Sheep's fescue)					xxxx	x		
<i>Festuca elatior</i> (Tall fescue)					x	xxxx		
<i>Cynosurus cristatus</i> (Crested dog's tail)					x	xxx		
<i>Avena fatua</i> (Wild oat)					x	xxx		
<i>Avena flavescens</i> (Golden oat)						xxxx		
<i>Diarrhiza arundinacea</i> (Reed)						xxxx	xx	
<i>Phleum pratense</i> (Timothy)						xxx	xx	
<i>Agropyrum repens</i> (Couch)						xxx		
<i>Agrostis stolonifera</i> (Creeping bent)						xxx	xxxx	xx
<i>Cynodon Dactylon</i> (Dog's tooth)						xx	xxxx	
WEEDS.								
<i>Plantago major</i> (Great plantain)					xxxx	xxxx	xxxx	
<i>Rumex crispus</i> (Curly or common dock)					xxx	xxxx	xxx	
<i>Atriplex hastata</i> (Orache)						x	xxxx	xxxx
<i>Chenopodium rubrum</i> (Red goosefoot)							xxxx	xxxx

northwards. This corresponds principally with the period of pollination of the grasses, which are the most important cause of hay-fever in the British Isles. Unlike other parts of the world, trees and weeds are relatively unimportant factors. In the accompanying table the relative order and approximate dates of pollination of the commoner British hay-fever-producing plants are set out. The same pollens apply largely to the Continent of Europe as well.

Common flowers and cereals are popularly thought to be causes of hay-fever symptoms. Corn, wheat, oats, rice, barley and rye produce large, moist, heavy pollen granules which cannot be carried far by wind, so they can produce symptoms only in sensitised individuals who come into very close contact with them. Flowers are insect-pollinated, and produce only small quantities of a large, sticky, spiculated pollen, so that they can only be a factor in the causation of symptoms when the patient is brought into intimate contact with the plants either by occupation, in the adornment of the home, or by direct inhalation, as in actually smelling the flowers.

Factors Influencing the Intensity of Symptoms.

The relative abundance of pollen granules each year is influenced by such factors as wet weather early in the season, which increases the growth of plant life; by dry and sunny weather later in the season, which allows the free liberation of the pollen; and by a moderate to a high wind, giving the pollen a greater spread. During the pollination season sunshine is the greatest stimulus to pollen production, as a temperature of 75° F. is necessary to open the flowers and liberate the pollen. In the season this temperature is generally reached between the hours of six and eight a.m., and this explains why symptoms are generally most pronounced at that time. Below 60° F. all pollination ceases, and rainfall practically entirely clears the air of pollen, due to a settling down of the pollen clouds as well as to a lessened production. As pollen also tends to settle out of still air, a wind is necessary to bring the pollen into contact with the sufferer's mucous membranes. The initial symptoms generally commence after some massive exposure, such as occurs during a car ride in the country on a sunny, windy day, or during a picnic in a pollen-laden atmosphere, since larger amounts of pollen are encountered in such a way in a given time.

Ætiology.

From the preceding facts it will be evident that the relative frequency of hay-fever in different countries is a matter of botanical rather than of racial difference. Given an abundance of a specific pollen-exciting agent in the atmosphere, probably the factor which plays the most important rôle in determining the onset of symptoms is an hereditary allergic predisposition, symptoms occurring most frequently amongst families where other allergic diseases

occur, or in individuals manifesting other allergic symptoms. Males and females appear equally susceptible. The age-incidence is greatest from fifteen to forty years of age, and in this respect hay-fever differs from the other allergic diseases, all of which have their greatest age of onset during the first decade. The youngest case I have seen was in a baby of one year, the oldest in a woman of eighty years. Hay-fever is quite frequent during childhood, but it is rarely recognised, as it is commonly mis-diagnosed as a common cold.

Symptoms.

The onset of symptoms in mid-May is generally gradual, when a mild sensation of itching and burning at the inner canthi or within the naso-pharynx is experienced. The succeeding symptoms may be divided into two main categories, one oculo-naso-pharyngeal, the other asthmatic, whilst on occasions a pollen dermatitis has occurred.

The oculo-naso-pharyngeal symptoms are of a catarrhal nature, and consist of paroxysms of violent sneezing associated with a profuse stainless watery discharge from the nose which leave the patient utterly exhausted. Coincidentally there is a profuse congestion, watering and itching of the eyes, whilst the itching of the pharynx and the post-nasal discharge cause an irritative, hacking, non-productive cough. Chronic swelling of the pharyngeal mucosa in some cases leads to periodic deafness due to the obstruction of the Eustachian tubes. These symptoms are most marked in the early morning, when, as already described, the pollen content of the air reaches its maximum peak.

The asthmatic symptoms that affect about half the sufferers sooner or later, becoming more pronounced each season, are generally worst at night, though slight attacks may occur during the day. At first the asthmatic symptoms remain confined to the pollinating season and are due to pollen sensitisation in general, but in some cases, and in each successive year, the symptoms tend to last longer into the colder weather as new sensitisations arise. These include feathers, horsehair, dust, and orris root.

Diagnosis.

The diagnosis of hay-fever may be symptomatic or specific. In the symptomatic diagnosis the coryzal symptoms are more or less pathognomonic. The itching and burning are of variable

severity and associated with such meteorological factors as sunshine, rain and wind ; the sneezing is of a multiple paroxysmal nature ; the symptoms have a definite time of onset, periodicity and duration, and a surprisingly sudden termination ; there is a general absence of gross permanent pathological changes even in cases of long standing, and there is a strongly marked hereditary influence.

The specific diagnosis is accomplished by determining the effect of dry pollen or pollen extracts on the conjunctiva, nasal mucous membrane or skin, or, less scientifically, by noting the effect of a change of environment. The most common difficulty in differentiation is caused by the common cold, but if one remembers its infectious, feverish, and not so definitely seasonal nature, the comparative insignificance of the sneezing, and the red inflammatory mucous membranes of the cold, with their neutrophilic discharge, compared with the pale boggy membranes of the hay-fever sufferer, with their eosinophilic discharge, the two conditions can be rapidly distinguished.

Popular Fallacies.

From the foregoing remarks it will be realised that, contrary to popular opinion, hay-fever is not a contagious disease, and not all people are equally susceptible. It is not a nervous manifestation, nor a disease of nervous people, nor solely of the upper intelligent classes. It is not a trivial affection of the nose and eyes caused by goldenrod or roses, but commonly an hereditary constitutional disease due generally to wind-borne pollen, with frequent serious and distressing complications. It cannot be cured by a nasal operation. Though spontaneous cures do occur, they have little to do with those popular epochs of life—the multiples of seven.

Treatment.

The successful treatment of seasonal hay-fever depends rather upon the recognition of all possible specific causes than upon the detection of infections or structural alterations in the nose. No doubt the presence of the latter tend to accentuate hay-fever symptoms when they occur, but if all the specific causes are recognised and measures taken to render them void, the patient should be just as immune to symptoms in the summer as he is in the winter. In the absence of surgical intervention, it is remarkable the number of years that patients can suffer from hay-fever symptoms without any infection developing.

The specific causes of hay-fever with which we are concerned include not only the pollens of the locality, but usually also house dust, animal emanations and orris root, the basis of all common toilet preparations. If the patient is sensitive to these latter inhalants, their removal or substitution is essential, or desensitisation is necessary. Dust may be added to the stock pollen solution when the injections are begun. Orris-root-free cosmetics may be obtained specially from Messrs. Boutalls, Ltd., Chemists, 150, Southampton Row, London, W.C.1 ; for face powders are common irritants in women hay-fever sufferers, and soon a vicious circle is established ; the greater the sneezing the more powdering of the nose is necessary, which in turn induces more sneezing. Feather pillows should be removed, and the best Java kapok substituted. The recognition of these other inhalant sensitisations frequently makes the difference between a failure and a successful treatment.

For those who cannot afford the time and expense of a prolonged change of environment in order to avoid the pollens to which they are sensitive during the pollination season, desensitisation is a necessary therapeutic procedure. Once symptoms have commenced one is forced to undertake *co-seasonal* treatment, and a satisfactory course can be put through by giving injections of increasing doses of pollen solution each two hours from 8 a.m. to 8 p.m. In known cases the *pre-seasonal* method is that usually adopted, the course of injections commencing about the beginning of March, and continuing weekly until the beginning of June. Once the maximum dosage has been reached it is often advantageous to continue the treatment *perennially*. In such a case one injection of the maximum dose reached is given each month, and the patient is kept permanently immune for about three years, when very frequently all sensitivity is lost. This latter course is preferable to the pre-seasonal treatment, as the number of injections and visits is lessened, the treatment is less likely to be interrupted by illness or vacation, the results are invariably better, and a clinical cure can be established in a much shorter time.

The solutions of the common British pollens for testing and treatment may be obtained from Messrs. Duncan, Flockhart & Co., of 155, Farringdon Road, London, E.C.1. Skin tests are carried out with the common pollens of the district, and the solution for treatment should contain all the pollens to which the patient reacts. In the British Isles they are practically only grasses, though in

special circumstances isolated reactions occur to trees and weeds. In the choice of the pollens to employ it is wise to remember : (a) that there does not appear any cross protection amongst members of the tree group ; (b) that there is a certain amount of protection amongst members of the grass group ; and (c) that group protection is the rule amongst members of the weed groups. In other words, should a specific reaction to a tree occur, that tree solution must be included ; should several reactions to grasses occur, though two or three mixed together may be sufficient to mitigate the symptoms, the best results will be obtained when all are included ; should a reaction occur to a member of the chenopod or dock group, then a solution prepared from any member of the same family will do for desensitisation. The most frequent reactions amongst the grasses occur in Yorkshire Fog (*Holcus lanatus*), the meadow grasses (*Poa annua*, *pratensis* and *trivialis*), the Fescues (*Festuca ovina* and *elatior*) and to Timothy (*Phleum pratense*).

The maximum concentration of pollen supplied by the manufacturing houses is about a 5 per cent. solution, or 50,000 units of pollen per cubic centimetre. According to the relative positivity of the various skin reactions, different quantities of the concentrated pollen solutions are mixed together and labelled "strength A." A dilution fluid is prepared as follows (or may be obtained from Messrs. Duncan, Flockhart & Co.). A buffered salt solution is made as follows :

Sodium bicarbonate	2.7 grams
Sodium chloride	5 grams
Distilled water	1,000 c.c.

This is sterilised by Berkfeld filtration, as the bicarbonate solution cannot be boiled. The final diluting fluid is composed of :

Sterile glycerin	50 per cent.
Sterile buffered salt solution	50 per cent.

The first dilution, "strength B," is made by taking one cubic centimetre of the concentrated "strength A" and adding nine cubic centimetres of the diluting fluid. The second dilution, "strength C," is made by taking one cubic centimetre of "strength B" and adding nine cubic centimetres of diluting fluid. One-tenth of a cubic centimetre of "strength C" is a good initial dose if given subcutaneously, containing as it does about fifty pollen units,

and the dose is doubled twice a week: 0.1, 0.2, 0.4, 0.8 c.c. "Strength B" is commenced during the third week, and similar amounts are given at five- to seven-day intervals. "Strength A" is begun about the seventh week, and the dosage at weekly intervals should be 0.1, 0.15, 0.2, 0.3, 0.4, 0.5 c.c., etc. Once the season has commenced, say, after June 1st, it is unwise to increase the dose any further, as the patient is absorbing large quantities of pollen from the air each day. It is rarely necessary to continue the injections after the middle of June.

Should any general symptoms ensue after an injection, these can be cut short by five minims of the one-in-a-thousand adrenaline chloride solution hypodermically. If a large local erythema with itching and swelling follows any injection, the same dose should be repeated next time rather than increasing it according to the schedule. The second time the reaction is not so great. It is often wise to give the larger doses, especially if accompanied by any discomfort, in two halves, one injected into each arm. After the larger doses it is essential to keep the patient under observation for at least a quarter of an hour to watch the development of the wheal in order to forestall the possibility of a constitutional reaction. This may occur when too large an increase is made over the former dose, when changing from one strength to a fresher and more concentrated one, or from the introduction of the solution directly into a vein. The longer the reaction is delayed the less severe is it likely to be.

These injections may be continued over a period of years without leading to any functional or organic disturbances. Two-thirds of the cases derive great benefit immediately from the treatment according to the thoroughness with which it is carried out. The common causes of failure include incorrect diagnosis; improper treatment due to impotent extracts, insufficient injections or inadequate dosage; failure to recognise multiple sensitisations to factors other than pollens; and seasonal variations in the pollen crop leading to an extreme abundance of pollen at a given time.

Besides the preventive measures outlined, there are many palliative measures that bring great relief to sufferers during the season. The eyes can be relieved by the wearing of dark glasses, which cut out the glare and diminish the amount of pollen coming into contact with the conjunctiva. The itching and burning of

the conjunctival and nasal mucous membranes can be relieved by the following ointment :

Benzocaine	1 drachm
Ephedrine hydrochloride	10 grains
Liq. adrenal. hydrochlor. B.P.	1 drachm
Paraffin moll. alb.	to 2 oz.

Internally, ephedrine hydrochloride in doses of half to three-quarter grain orally, night and morning, is sufficient to control the spasms, but in acute exacerbations the hypodermic injection of adrenaline is often necessary. Hydrochloric acid has a beneficial effect if given internally ; this is best given in tablet form, one " Aklorep " tablet three times a day immediately before meals. (These are made by Roberts & Co., of 76, New Bond Street, London, W.1.)

Inhalations of carbon dioxide gas through a " Sparklet " apparatus, as suggested by Hurst, frequently lead to an immediate cessation of nasal symptoms. Cauterisation or ionisation of the nose do not generally lead to any permanent benefit, though at times useful as palliative measures. Vaccines and non-specific protein therapy are generally a waste of time and money.

Further particulars as to skin testing and its technique, pictures of the common British hay-fever-producing plants, as well as all recent literary references to hay-fever, may be found in " Recent Advances in Allergy," G. W. Bray (London : Messrs. J. & A. Churchill. 1934).

CHAPTER XXII.

THE EPILEPSIES IN CHILDHOOD

BY

W. G. WYLLIE, M.D., F.R.C.P.

Physician to Out-patients, Hospital for Sick Children, Great Ormond Street,
London ; Assistant Physician, Hospital for Epilepsy and Paralysis, Maida Vale,
London.

It is customary to make arbitrary distinctions between different types of epilepsy : the essential or idiopathic, and the symptomatic. Infantile convulsions, further, are by many set apart as a separate species. Actually, the fits in any of these groups are all of the same character, and are all epileptic. An epileptic fit, as Kinnier Wilson has said, is nothing else than a symptom: "no such disease as epilepsy exists, or can possibly exist." The nature of the nervous element, or the physio-chemical changes in cortical cells that lead up to the discharge of a convulsion, are beyond our knowledge. Inciting factors, however, are numerous, and may be vascular, inflammatory, toxic, metabolic, traumatic, and neoplastic in origin. The prefix "essential" or "idiopathic" has no meaning beyond signifying the absence of any apparent inciting cause. It would, therefore, be preferable to employ the collective term—the *epilepsies*.

In childhood the epilepsies can with benefit be divided chronologically into three groups : (a) those which occur at birth and in the neo-natal period ; (b) epileptic fits in infancy ; and (c) from the end of the second year onwards.

(a) *Convulsions in the newborn due to birth trauma*.—At birth intracranial hæmorrhage is most apt to occur in cases of prolonged and difficult labour, pre-maturity and precipitate delivery. Venous congestion from asphyxia is the chief cause of bleeding, which may be intracerebral, subdural, subarachnoid or intraventricular. The bleeding may also be due to sudden alterations of intracranial pressure as in precipitate delivery, or to vascular immaturity in the premature. Frequent convulsions at birth signify extensive

cerebral injury, and the infant seldom survives. Retinal hæmorrhages are often present. A considerable unilateral subdural hæmorrhage, by pressure atrophy of the subjacent cortex, may give rise to convulsions and a contralateral hemiplegia, which do not make their appearance until several weeks after birth. A small percentage of children with diplegias of ante-natal and natal origin have convulsions, with onset at any time during childhood or in later life. When convulsions occur at or soon after birth, lumbar puncture is a useful diagnostic aid to determine the existence of meningeal bleeding. Little success can be expected from cranial operations, as only under the most favourable circumstances can more than a small portion of the extravasated blood be removed. Treatment must largely be symptomatic, taking into account the possibility of paralysis and amentia, as well as of convulsions following intracranial damage at birth.

(b) *In infancy*.—(1) Occasionally, in apparently healthy infants of normal birth, convulsions occur within the first few months, and sometimes as early as the first week. Dr. Thomson referred to these fits as "*idiopathic convulsions*" of infancy. Breast and bottle-fed infants are affected, but boys more often than girls. At first the seizures are slight and of short duration, but increase in number and severity from day to day. Under suitable treatment the prognosis is good in most cases: the convulsive tendency is arrested and the integrity of the cerebral functions escapes injury. The best results are obtained by the use of chloral, in doses sufficient to render the infant very drowsy, but still able to suck. At first a dose of one or two grains, corresponding to the age of the infant in months, is given every two or three hours until the convulsions have ceased. The effect of the drug must be carefully watched. When the convulsions have ceased, the dosage of the drug is gradually decreased in quantity and frequency.

(2) In a somewhat similar group of cases convulsions commence in previously healthy infants towards the end of the first and in the second and third years. The onset, however, is abrupt, and the convulsions are severe and numerous from the start. Between the major seizures the infant is constantly twitching and not fully conscious, although feeding is possible. At the end of several days, or a week or two, when the fits have ceased, the presence is discovered of either a hemiplegia or amentia, or both. The pathology

of the condition is obscure, as it is rarely fatal. Strümpell, in 1884, referred to it as *sporadic encephalitis*, and maintained that a close analogy existed between these cases and polioencephalitis. This view has not been confirmed serologically, as the cerebro-spinal fluid gives no evidence of a virus infection. The possibility of a vascular lesion must be considered as one of the causes. In a case seen recently, which succumbed to a secondary pneumonia, thrombosis of one of the cerebral arteries was discovered. The prognosis is bad in these cases. Repeated doses of chloral provide the best means of overcoming the fits.

(3) When convulsions occur between the sixth month and the end of the second year, the possibility of a calcium deficiency must be considered. Obvious signs of rickets may be present, provided the blood phosphorus as well as the calcium is reduced. In other cases without skeletal deformities a flabby and atonic state of the muscles is often noticeable. Besides the convulsions, other manifestations of spasmophilia may be present, such as tetany and laryngeal stridor. The convulsions in these cases tend to occur at the time of the greatest seasonal incidence of rickets and tetany, that is, in the winter months.

Calcium is known to be a damper of nervous excitability, and a deficiency of the ionised quotient of the blood calcium facilitates the occurrence of convulsions. In cases of this nature estimations of the serum calcium commonly lie between 4 and 8 mgm. per 100 c.c., in place of the normal 10-11 mgm. per cent. Teething convulsions are thought to be of a similar nature. It does not follow, however, that in every case a low blood-calcium will give rise to convulsions. Some additional factor would seem to be necessary which is probably constitutional and of the nature of a nervous instability. In this group of cases treatment is attended by the most gratifying results. When the fits have been few in number, Radiostoleum, minims 5, three times daily, by its effects upon calcium absorption, will speedily effect a cure. The combination of tetany and convulsions is treated by the administration of calcium chloride, 15-30 grains, four-hourly for three days, and cod-liver oil and artificial sunlight for a longer period. Calcium chloride exerts its influence, not by the calcium, which is largely unabsorbed, but by the absorption of the chlorine portion, thereby producing a tendency to acidosis, which facilitates the ionisation of calcium.

(4) The influence of a constitutional instability of the nervous system has long been held to play a part in the ætiology of convulsions in some cases. It is not uncommon to obtain a history of convulsions in infancy in one or other parent, as well as in one or more of their children. Convulsions of this nature occur sometimes in association with sources of reflex irritation, such as colds, earache, gastro-intestinal disturbances, worms and other minor upsets of health. The blood calcium in the majority of these cases is normal. This constitutional tendency is further noticeable when one or more members of a family in infancy or early childhood have a convulsion at the commencement of a febrile attack, as in pneumonia, or in one of the infectious fevers.

In a similar group of cases convulsions occur during febrile disturbances, associated with a marked ketosis. The origin of the infection is most commonly tonsillar, and the attacks may be repeated several times during the latter half of infancy and the following two or three years. Vomiting is also a common symptom. The epileptic fits cannot be attributed to the effects of the ketosis, as neither experimentally in animals does acetone, except in massive dosage, produce convulsions, nor do they occur in children on a ketogenic diet. A diet too high in fats and low in carbohydrates has been blamed in these cases, but there is no evidence that hypoglycæmia is a cause of the fits. A more probable explanation in some cases is a tendency to alkalosis as a result of the fever, with a fall in the serum calcium. In the majority of the cases the convulsive tendency disappears during early childhood. Treatment is symptomatic. Foci of irritation, gastro-intestinal and tonsillar, must receive attention. For the fits themselves, bromides and chloral are given.

(5) The epileptic fit may be an early symptom of meningitis. Posterior basic meningitis has its greatest incidence in the first year of life. Early symptoms are listlessness, screaming, a bulging fontanelle, vomiting and convulsions; opisthotonos develops at a later stage. In tuberculous meningitis the greatest number of cases are in the second and third years, but the disease may occur as early as the third month. Convulsions are common in the terminal stages, but may be among the first definite symptoms. Twitchings, unilateral and bilateral convulsive movements in early infancy, may be due to congenital syphilis involving the brain and its meninges. Amentia is usually present. It must, however, be

remembered that minor fits, *petit mal*, in an otherwise normal child, may be the sole manifestation of congenital syphilis. The origin of the fit in some cases is suggested by a maternal history of miscarriages and stillbirths, or signs of neurosyphilis in the mother. In these conditions treatment must be mainly directed towards the primary disease.

(c) *In the years following, as well as during infancy, epileptic fits have many different causes.*—In some children of a type already described they occur during febrile bouts, such as pneumonia, tonsillitis, otitis media and the infectious fevers. A fit at the onset of an illness has little prognostic importance, whereas in any child which is seriously ill late convulsions are of bad omen, and a fatal termination is to be expected. Convulsions are a common symptom of the many forms of meningitis and encephalitis.

In some cases of migraine in the child, fits indistinguishable from *petit mal* occur at the height of the attack. The child has a lapse of consciousness or falls to the ground, with a change of colour, and remains stiff for a few minutes. A convulsion is more rare. The periodic syndrome constituting migraine in the child is composed of headache, commonly frontal and bilateral, abdominal pain, vomiting, and in many cases fever. Constipation and an excess of mucus are commonly present. Treatment consists in giving sod. bicarbonate, pulv. rhei and syrup of senna. Small doses of thyroid dried extract and of luminal often benefit the headaches. Lately a ketogenic diet has been advocated for the treatment of the migrainous patient.

In pyknolepsy, attacks in the nature of *petit mal* commence between the fourth and tenth years and cease at or before puberty. As many as a hundred or more may occur in the day. An attack consists of a lapse of consciousness, usually without spasm or falling, and with no subsequent mental confusion. Mental deterioration is said not to occur. An outstanding feature of the complaint is that bromides and luminal have no effect upon the frequency of the fits. The condition is a rare one. It is rash to give a good prognosis in a case of this nature, as it has happened not infrequently that a later appearance of major epileptic attacks has necessitated an alteration in the diagnosis.

In epilepsy of the "idiopathic" type, Gowers stated that one-eighth of all cases began during the first three years of life. Quite often the fits are in the form of *petit mal* up to the age of puberty,

when major attacks may have their first appearance. In children auræ of the special senses are rarely experienced. Epigastric and abdominal premonitions are much more common. A tendency to a regular periodicity is often noticed. Many children have the majority of their attacks during the night or in the waking hours of the morning. Mental deterioration is not necessarily a natural consequence of epilepsy. Many epileptics retain their mental powers unimpaired. Mental deficiency is more apt to occur when the fits commence in early childhood, but in many cases both the epilepsy and the mental defect are symptomatic of an abnormality of cerebral development. Salaam attacks and other posturing attacks, with bizarre movements of the arms, in young children are usually associated with mental defect. The epileptic seizure is often of the Jacksonian type in children, and it is quite erroneous to infer that a local cerebral lesion exists in such cases. Of children who have had infantile convulsions, roughly ten per cent. have epilepsy in later life.

In the treatment of idiopathic epilepsy no hard-and-fast rules can be laid down. Different cases respond best to different remedies. Luminal in tablet form is usually the most efficacious drug. For the infant, quarter of a grain three times daily is given; for the older child, grains a-half to one, twice daily. The bromides should also be tried, also sod-biborate, grains five, or urethane, grains five to ten thrice daily, for a child of seven to ten years. When fits have been frequent, syrup bromocarpinæ, drachms one to two, thrice daily, for a child of seven years, may be useful as a temporary measure when other drugs have failed. In some cases of *petit mal*, in otherwise normal children, a favourable response is obtained to ephedrine hydrochloride, grain a-quarter thrice daily, or grain a-half twice daily.

Great benefit has been claimed by some from the use of the ketogenic diet in epilepsy, while others have found it ineffective. An occasional case responds to this treatment, while others are made definitely worse. There is no means of telling beforehand which case is likely to benefit. In the young child the diet is difficult to arrange, as digestion may easily be upset. Generally speaking, carbohydrates are much reduced, and eggs, butter, fat bacon, fried fish, cream, top milk, cod-liver oil and vegetables cooked in butter, are given in liberal amounts. Acetone should be present in the urine.

In *status epilepticus*, when convulsions succeed one another so rapidly that consciousness is not recovered between attacks, the danger to life is due more to heart failure than to the fits themselves. The quickest and best method of treatment is to give a hypodermic injection of morphia, grains $1/12$, $1/16$, or $1/20$, according to age. Hyoscin hydrobromide, grain $1/500$, is sometimes given at the same time. Chloroform inhalations may be useful until the drug takes effect. Rectal injections of chloral and of paraldehyde are of use when the convulsions are less frequent, but are very apt to be ejected during the violent peristalsis which occurs during the fit.

CHAPTER XXIII.

FLAT FOOT

BY

E. P. BROCKMAN, M.Ch., F.R.C.S.

Orthopædic Surgeon, Westminster Hospital.

A **FLAT** foot is commonly understood to mean a foot in which the longitudinal arches have collapsed. The normal foot has certain well-defined arches which are produced and maintained by the action of the muscles of the leg and foot. Should for any reason this muscular power fail, these arches collapse, and the condition known as flat foot develops. On careful examination of any flat foot it will be seen that this lowering of the arch is really an abduction and eversion of the forefoot at the midtarsal joint, which thus allows the head of the astragalus to come nearer the ground. It will be noticed that when the forefoot is adducted and inverted the flattening of the arch disappears. For practical purposes there are five types of flat feet which need to be recognised.

Infantile Flat Foot.

A normal child when it first commences to stand always does so with its feet flat. It needs to be realised that such a flat foot is only a stage in its normal development, for any amount of time and money has been wasted in the treatment of these children. The reason that a child when it first assumes the erect posture has flat feet is that its weight is too great for its muscular power, and also because it has not at this stage learnt how to use its muscles to the best mechanical advantage. As the child learns how to stand and balance itself, these muscles begin to work properly and the arches of the feet to develop. It usually takes several months before this occurs, or it may even be a year or two before the foot assumes what we may regard as a normal appearance. Children who have had rickets, or whose feeding has been unsatisfactory, usually take much longer to develop a normal foot, because

their muscles are flabby. And a poor musculature of this nature may take several years to develop a proper tone. The parents of these children are apt to worry very much about their flat feet, but if it is explained to them that it is a normal thing for children to have such feet when they first begin to stand, their minds can generally be set at rest.

No active measures are called for, but it is useful to wedge the inner side of the sole and heel of the shoes a quarter of an inch to relieve any strain on the ligaments of the foot, and also with the idea of assisting the muscles to carry out their function. Massage to such children's feet is largely waste of time, for it will not hasten the development of their muscular power.

Flat Foot due to a Short Tendo Achillis.

If a child who is otherwise healthy continues after the age of four or five to have flat feet we must then look for some other reason. The most common cause for a persistent flat foot in a young child is a congenital shortening of the tendo achillis. When this tendon is contracted, the child, in order to get the foot properly in contact with the ground, must needs abduct and evert the fore-foot. Otherwise it would have to walk on its toes. Such a condition is more often seen in girls than in boys. We need to recognise such an abnormality, for if we do so we can save the child being condemned for years to wear ugly and heavy surgical boots with all sorts of contrivances added to them, and also can avoid perhaps many years of quite useless exercises. When a foot in which a shortened tendo achillis is present is held in the normal position, with the longitudinal arches restored, it will be found that the foot cannot be dorsiflexed above the right angle, as it should be in a normal foot.

If the shortening of this tendon is only of a minor degree, it can be compensated for by raising the heel of the shoe by a third of an inch, which will be sufficient to allow the forefoot to return to the mid position. Should the degree of contraction be very marked, it interferes with the normal action of the foot to a considerable extent, and the child will stand with its feet very much everted, and it will also have great difficulty in running at a pace sufficiently fast to enable it to keep up with other children. If the condition is not identified before the child goes to school, he will often say that he is not able to run fast enough to ever be

able to win a race, and that long walks tire his feet. This, of course, is only natural, for with an everted foot he has little of the natural spring which should be present. Under these conditions it is advisable to elongate the shortened tendon by the subcutaneous method, and afterwards to fix the foot at a right angle, with the forefoot slightly adducted, for a period of six weeks. When the plaster is removed, massage and exercises are necessary over a long period to teach the child how to use its muscles, for we have to remember that up to this time its muscles have been always working at a disadvantage. The parents need to be warned after this operation that such a child will take many months, or even a year, before it will be able to run about and play games like other children. For several months they tend to be clumsy in their walking, and to do so on their heels.

Adolescent Flat Foot.

This type of flat foot is seen most often in the out-patient department of a hospital, but it may occur in any class of society. It is also known as spasmodic flat foot, and consists of a painful rigid foot, associated with spasm of the extensor longus digitorum and peronei muscles. The usual history is that some months, or even a year, previous the patient left school and went to work at a job which entailed many hours of standing. All went well to commence with, but after some weeks his feet were tired at the end of the day. This continued, and in time got worse, until in addition to being tired, they started to become painful, either on the inner side over the astragalo-scapoid joint, or on the outer side over the calcaneo-cuboid joint. Rest at night relieved the symptoms, but very rapidly the pain became continuous, and the patient had the greatest difficulty in walking about. When examined, such a foot is found to be held rigidly abducted and everted. Inversion and plantar flexion cannot be carried out either actively or passively, and any attempts to do so give rise to pain. The tendons of the extensor longus digitorum and the two peronei stand out like bow-strings, and if stretched are very painful. Many theories have been advanced to explain this condition, including the suggestion that it is an arthritis of the tarsal joints, the seat of infection being the tonsils. The true explanation probably is that the child starts off with good feet, but that as the result of hours of unaccustomed standing, often in the same

position, the muscular power gives out and the muscles are no longer capable of supporting the foot. The strain then falls on the ligaments of the foot, which were never intended to take such strain, their function being solely to connect up the bones of the foot. As the result of having to take this strain, these ligaments become inflamed and stretched, with the result that the articular surfaces of the bones do not fit accurately together. As a consequence of this a synovitis, and then an arthritis, is set up, and the extensor longus digitorum and peronei go into a protective spasm, which, if it lasts any length of time, causes an organic shortening of the muscles and tendons. The treatment usually advocated for spasmodic flat foot has the disadvantage that the patient must give up work for at least three months, and thus lose employment. Moreover, the results of the classical treatment of this condition, wrenching and fixation in plaster, are not such as warrant a prolonged period of inactivity. If the condition has been present for any length of time, the patient should have his feet manipulated under an anæsthetic to restore mobility. Afterwards he is kept in bed and given radiant heat, massage and exercises, to maintain the movement obtained by manipulation. At the end of a fortnight the patient may be got up with wedged shoes, and is provided with short outside irons and inside T straps. He should continue with exercises and the use of instruments for at least six months, but he can meanwhile resume his work. Treated in this fashion, early examples of this condition obtain mobile feet which cause no further trouble. When a spasmodic flat foot has been present for many months it is doubtful whether the foot can ever be restored to its former appearance. Forcible manipulation, even with a Thomas's wrench, generally fails to alter the condition of the foot to any appreciable extent, and the contracted tendons cannot be stretched out. These tendons are sometimes tenotomised before the foot is wrenched and put in plaster, but when the plaster is removed they are found to have joined up as if nothing had been done to them, and the appearance of the foot is similar to what it was before treatment started. It is as well to appreciate that this method of treatment fails to cure the condition, and that when a spasmodic flat foot has been present for any length of time the rigidity is permanent, and we can only hope to relieve symptoms. This can be achieved by providing the patient with an outside iron and an inside T strap, and wedged shoes to be worn for six to nine

months. His symptoms will settle down very rapidly, and the final condition of the foot is as good, if not better, than it would have been after forcible manipulation, with the avoidance of enforced loss of work for two to three months.

Flat Foot in Adults.

In any community a very large number of individuals have feet which are flat, but which, giving rise to no symptoms, do not call for treatment. It is only when these feet are strained, either by overwork or ill-health, that symptoms of tiredness or pain are complained about. Just as in the younger patients, the muscles cease to do their work properly, and the ligaments being stretched become painful and tender and the movements of the tarsal joints limited. It is this limitation of movement in the adult flat foot which causes pain. In other words, an adult flat foot, provided it is mobile, gives rise to no disability. Only when, in addition to being flat, it starts to become rigid does trouble commence. Such feet are seen in men and women alike, and our treatment should aim at making these feet mobile again, and having obtained this mobility, their joints must be kept free and the muscles restored to full function by suitable exercises. Radiant heat, massage and forced movements of the tarsal joints in mild cases will do all that is required. But in feet which have had symptoms for a long time, forcible manipulation under an anæsthetic is necessary before the masseuse can hope to do any good.

At times one encounters rigid flat feet in adults in which there is little if any movement in any of the tarsal joints, and in these patients it is wise not to try and force them, but to make them as comfortable as possible with suitable shoes. Metal supports and valgus insoles will enable a number of patients to carry on in comfort, but if the feet can be rendered painless by other means, so much the better.

Painful flat feet in women are very often the result of wearing unsuitable shoes with too low a heel, which causes a strain to be thrown on the longitudinal arches, and gives rise to pain. Very few women are capable of walking, except in the country, in low heels. Their feet are so constructed that a moderately high heel is essential, and the relief of painful flat feet in young women can usually be obtained by attention to their shoes.

It will be found that a number of patients with painful flat

feet do not respond to the ordinary methods of treatment, and examination shows that their symptoms are due to tenderness in the plantar fascia, muscles and ligaments. This type of foot is the result of infection. There are two classes of this acute, painful, flat foot. The first, which is quite common, follows an attack of influenza or tonsillitis three or four weeks previously. It comes on suddenly with little or no swelling, and responds to treatment by radiant heat and massage very quickly.

The second type of acute flat foot is uncommon, and is the result of an infection of the genito-urinary tract by the gonococcus. Any acute flat foot with a lot of pain and swelling of the tissues of the foot, especially in a young man adult, for which no obvious cause can be found, should always be regarded with suspicion, and the urinary tract examined. With treatment of the primary focus the condition of the feet rapidly subsides if these are rested during the acute stage. Occasionally a chronic infection of the prostate will also cause a painful inflammation of the tissues of the sole of the foot.

CHAPTER XXIV.

THE MODERN TREATMENT OF PYORRHŒA

BY

A. T. PITTS, D.S.O., M.R.C.S., L.R.C.P., L.D.S.

Dental Surgeon to the Royal Dental Hospital and the Middlesex Hospital ;
Examiner in Dental Surgery to the Royal College of Surgeons.

THE term "pyorrhœa alveolaris" in a broad sense may be taken to mean a chronic general infection of the supporting tissues of the teeth, leading to their eventual destruction. The implication in the term that a discharge of pus from the gum margins is the most important feature of the disease is of doubtful truth. At some stage in most cases there is a discharge of pus, but it is not invariable, nor is it necessarily the most significant feature. Perhaps the following definition* which the writer has given elsewhere, might command a fairly general, though not unanimous, approval: "Chronic general periodontitis (pyorrhœa alveolaris) is a disease characterised by a progressive inflammation and ulceration commencing at the gum margin and invading the periodontal membrane. Concurrently with the destruction of the periodontal membrane there is a rarefaction of the alveolar bone. As the supporting tissues of the teeth become destroyed the teeth become gradually looser. At some stage of the disease there is usually, though not invariably, a discharge of pus from the pockets formed by the destruction of the periodontal membrane. Secondary changes of a necrotic or regressive character affect the cementum, dentine and pulp. In some cases the bone changes are of a progressive, rarefactive character throughout, but in other cases rarefaction may be replaced by sclerosis or production of bone with fibrosis of the gum, so that the teeth remain firmly implanted until a relatively late stage." Such a definition describes the common

* Bennett's "Science and Practice of Dental Surgery," 2nd ed., 1931, vol. ii, chap. xxx.

sequence of the clinical features. One point of clinical interest emerges, which is that two main types of pyorrhœa can be recognised. In one the pockets are deep and the progress of the disease is rapid. There is a good deal of tartar on the teeth. The gums are hyperæmic and dark in colour. They bleed readily, and are often tender. The patients are often young adults with narrow, slender jaws. There is often a history of nasal obstruction and mouth-breathing. In this type we may say that there is little sign of tissue resistance to the infection, and general symptoms of toxæmia are not infrequently present. In the second type there is a fibrosis of the gums which proceeds concurrently with the bone destruction, so that deep pockets are not formed. The gums may be normal in colour and free from tenderness. There is not much tartar present. The relative absence of pockets means that food and bacterial stagnation do not occur. The jaws are large, and the dental arches well formed. The patients have a vigorous habit of chewing, and there is usually a good deal of wear of the teeth. Such patients may be in excellent health, with an entire absence of any general symptoms which could be referred to the oral condition. But in the end, though this may be long delayed, the bone destruction will outstrip the fibrosis of the gums, and pockets will be formed which permit of stagnation of food debris and bacteria. It is permissible to regard the fibrosis of the gums and sclerosis of bone as signs of tissue resistance. Clinically, the prognosis is good, and little treatment is required.

It is impossible to discuss the treatment of pyorrhœa without some reference to its ætiology. Much confusion exists, and theories abound, all of which have some plausibility, but lack convincing proof. Some authorities hold strongly that pyorrhœa is local in origin. There is much to be said for this point of view. Yet it overlooks certain aspects which suggest some general factor. All conditions which favour food stagnation undoubtedly predispose to pyorrhœa. Thus mouth-breathing, irregularities of the teeth, dietetic factors, spacing due to loss of teeth, defective fillings or crowns, and ill-fitting dentures—all of these may favour the onset of the disease. Yet it occurs in patients in whom none of these factors is present, while, conversely, local conditions unfavourable to oral hygiene may be compatible with a good condition of the gums. Other authorities would find the explanation in some general metabolic disturbance, which, co-operating with

some local condition, determines the onset of pyorrhœa. The views of F. W. Broderick have attracted considerable interest. He believes that the disease is primarily due to endocrine disturbance, which upsets the balance of calcium metabolism in the direction of lime saturation, and brings about a chronic alkalosis. This increases the calcium content of the saliva, and causes a diminution of the CO_2 content of the blood, leading to a deposition of calculus around the necks of the teeth, which in its turn is the actual exciting cause of the gingival lesions.

It is not possible to discuss further this vexed question, but enough has been said to make it clear that the treatment of pyorrhœa is to some extent empirical and directed to the amelioration of symptoms rather than a rational attack based on a proven ætiology.

Treatment.

It must be premised that cure in the sense of a restoration to the normal is not yet possible. Since the essential lesion is a destruction of bone and periodontal membrane, a restoration to the normal would imply a reformation of bone and a re-attachment of the periodontal membrane. There is no evidence that this is possible. This is not to say that with a more accurate knowledge of ætiology and pathology a method of treatment may not be devised which will make such a complete cure possible; but at the present time we know of no treatment which can accomplish such a regeneration of tissue. It is true that a gingivitis in which the supporting tissues are not affected may be completely curable without permanent loss of tissues. Since many writers hold that pyorrhœa starts as a gingivitis, in this sense it might be said that a restoration to the normal might occur. But the writer has always felt that this was an unfair attitude. Assuming that in every case of pyorrhœa there has been an antecedent and initial gingivitis, yet the converse does not hold good that every case of gingivitis is the prelude to pyorrhœa, for many cases of gingivitis occur which do not lead to pyorrhœa. It is easy to claim to cure pyorrhœa in this restricted sense, but it is essentially a deception, and, in contrast with more modest claims, suggests the possession of some form of therapy not generally known. But the writer must hasten to say that such claims usually proceed from loose thinking rather than from any deliberate intention to deceive.

In the present light of our knowledge the word "cure" as applied

to the treatment of pyorrhœa means a stabilisation, as permanent as possible, of the destruction of the supporting tissues of the teeth at the level they have arrived at when treatment commences. The disease may be arrested therefore, and since the term "cured" is often used in this sense, it is legitimate to apply it to the treatment of pyorrhœa, provided that its limitations be clearly understood.

There is a vast and ever-increasing literature devoted to the treatment of pyorrhœa which reveals more clearly than anything else the inadequacies of our knowledge. It is a sound procedure to examine all methods of treatment of a disease in the light of general principles. When so examined it will be found that many new forms of treatment, for all their apparent novelty, are only fresh expressions of old principles.

The various methods of treating pyorrhœa can be grouped under such headings as mechanical, surgical, medicinal, electro-therapeutical and immunological. It is safe to say that all the many methods which have been advocated fall under one or other of these headings. A simpler grouping would be those which destroy infection and those which promote drainage. A still further division of great importance is treatment carried out by the dentist and that carried out by the patient. Each is complementary to the other, and the absence of co-operation by the patient is fatal to success. It should also be emphasised that no one method is likely to be successful alone; it is the combination of various methods which is all-important.

Mechanical Treatment.

The first thing which should be done in every case of pyorrhœa is to scale the teeth thoroughly. It is essential to remove all tartar from the teeth, especially those particles which adhere to the roots, and are thus below the gum margins. If this is not done, all other treatment is a waste of time and money. This is far from easy, and may necessitate several visits. A mere hasty removal of the obvious masses of tartar followed by a polishing of the teeth is useless. To be effective it must be a carefully planned procedure, which demands patience and a delicate tactile sense.

Massage of the Gums.

Following the scaling of the teeth, a course of massage of the gums should always be instituted. Rubber vibrators which can be used in the dental engine have been devised, but in the writer's

view massage is essentially a form of treatment to be carried out regularly by the patient. A useful adjuvant is glycerine and tannic acid. The procedure is to wrap a piece of muslin round the finger and dip it in the glycerine and tannic acid. The gums should be vigorously rubbed, both transversely and vertically and on both surfaces. This should be done for a few minutes twice a day. A period of about three weeks is sufficient for the use of glycerine and tannic acid. It may then be replaced by a solution of salt and water—a teaspoonful of salt to a tumbler of water. This should form a permanent part of the hygiene of the mouth as practised by the patient. Massage acts by inducing a circulation of blood in the tissues, and promotes fibrosis of the gums. It is surprising how quickly it brings about an improvement.—In two or three weeks it is quite common to find that the tenderness and tendency to bleed readily have disappeared; the gums lose their dark, congested appearance, while their shrinkage is shown frequently by the presence of fragments of tartar deeply placed, which have become exposed like rocks by a receding tide.

Treatment by Drugs.

The local action of drugs depends on their action as caustics, disinfectants, or astringents. Innumerable drugs have been advocated, and good results have been claimed for all of them. There is nothing to suggest that any drug has a specific action. Creosote and alcohol in equal parts and chromic acid, 5 per cent. strength, may be taken as representative of caustics. Their usefulness, however, is evanescent. So long as pockets remain, stagnation must occur. Still, they have a place in the treatment of pyorrhœa.

The disinfectants are even more numerous. Hydrogen peroxide acts as a mechanical agent, through the bubbles of oxygen removing debris, rather than as a disinfectant. Its use, however, is considerable. Most dentists have compressed air laid on in their surgeries, and it is a useful procedure to spray round the mouth and flush out the pockets with peroxide on each visit. Iodine, mercurochrome and lysol are a few disinfectants which may be mentioned. Since spirochætes in combination with the fusiform bacillus are often present in the pockets, the use of a preparation containing arsenic has been advocated by Kritchevsky and Séguin. But evidence that these organisms in symbiosis are the cause of

pyorrhœa is lacking. While liquor arsenicalis may be useful, there is nothing to show that it acts as a specific. Ipecacuanha, or its active principle, emetine, has also been employed on the assumption that the endamœba buccalis, which is often present, is ætiologically significant. Here, again, there is nothing to show that this is so. Specific therapy, therefore, does not exist, in spite of extravagant claims which are sometimes made for various remedies. Astringent drugs like copper sulphate and zinc chloride are useful in the early stage in order to promote shrinkage of the inflamed gums.

Mouth-Washes.

It is perhaps not an exaggeration to say that the most important ingredient in most mouth-washes is the water. Their main purpose is to flush out the mouth and to remove debris. Claims that this or that drug will destroy bacteria found in the mouth may be true of experiments *in vitro*, but it is a fallacy to imagine that any antiseptic can be used in the mouth in such a strength that it will reduce the bacterial flora to any appreciable degree. Experiments which have been carried out show that any reduction is temporary and quickly made up. When it is remembered that in pyorrhœa there are irregular pockets round the teeth, lined with infected tissue, it will be obvious that the antiseptics used in a mouth-wash can only play a minor part in the treatment of pyorrhœa. But few patients will believe this; so that if only to inspire confidence, a mouth-wash containing some antiseptic is a useful measure. Astringent mouth-washes are useful however. A pleasant and effective example is Layloris, a Canadian preparation. Hydrogen peroxide (five vols.) is useful, as the mechanical flushing action is increased by the effervescence of the nascent oxygen. An ingenious apparatus called the Hygienator has recently been put on the market which embodies the principle of the "Sparklet" syphon. A suitable solution containing CO₂ under pressure, to which hydrogen peroxide can be added, can easily be made up, and the fluid can be sprayed round the mouth under considerable pressure.

Surgical Treatment.

Since all tissue above the level where bone and periodontal membrane are still intact, adds nothing to the stability of the teeth, and merely allows stagnation, it would seem a rational procedure

to excise this tissue and thus obliterate the pockets. For many years this has been a common procedure, giving excellent results. It should only be done after preliminary treatment to reduce inflammation and to get the tissue into as healthy a condition as possible. The operation can be performed under a local anæsthetic. Small and very sharp scalpels must be used, and various useful types have been devised.* Recently H. Stones has advocated gingivectomy on a more extensive scale. He excises a strip of tissue on both labial and lingual aspects down to the level of the bone round all the teeth affected. This is a lengthy procedure which, according to Stones, may take over an hour to perform. It can be done under a local anæsthetic, but obviously is much more severe than the excision of the pockets of a few teeth at a time, chiefly in the incisor and canine region. Good results are claimed for this treatment, which has the merit of being based on a sound pathology. But it must be emphasised that it does not replace other methods, such as massage and scaling, nor is it applicable to every type of case. Some operators prefer the cautery to the scalpel. Even more elaborate methods have been advocated in America, involving reflection of the gum and curettage of the bone, but the writer remains sceptical of their advantage over methods less pretentious and severe.

General Treatment.

It is highly probable that in many cases of pyorrhœa there are general factors of importance. But too little is known to enable us to use a therapy of a general character with much certainty. It has been argued that the local condition may be secondary to intestinal stasis, and that treatment devoted to the intestinal condition, such as the regular use of liquid paraffin, would be beneficial. Gottlieb recommends the use of Fowler's solution internally, commencing with five drops once daily for five days, and gradually working up by successive increases of five drops every five days up to thirty drops daily. The dose is then diminished gradually to five drops again. He believes that arsenic stimulates the growth of bone. F. W. Broderick, whose views have attracted much attention, believes that pyorrhœa is essentially a local reaction to a condition of alkalosis. He advocates the use internally of ammonium chloride. About two grains should be taken before

breakfast. After a few days the reaction of the saliva should be tested and the dose increased or diminished until normal salivary pH is reached.

Electrotherapy.

The action of drugs may be increased by ionic medication. Those who practise this method are enthusiastic in their belief in its value. It is claimed that a deeper penetration of the drug into the infected tissue of the pockets can be obtained by ionisation. Such drugs as zinc chloride, tincture of iodine and cuprol can be used. A few teeth should be treated at a time with a current ranging from one to four milliampères. The electrode should be held in contact in one spot from one to three minutes.

Vaccine Therapy.

Many organisms are found in pyorrhœa pockets, but none of them fulfils the necessary criteria to be regarded as the specific cause. From this point of view vaccine therapy is empirical. Since, however, the streptococci present are in all probability the organisms responsible for the general symptoms which may be caused by a focal infection in the mouth, a vaccine may sometimes benefit the general health. But whether vaccines do any good locally is another matter. The general opinion at the present time is that vaccine therapy has little if any value in the local treatment of pyorrhœa.

The Extraction of Teeth.

No account of the treatment of pyorrhœa, however brief, would be complete without some reference to the extraction of teeth as part of the treatment. Space does not permit of a discussion of the points which will influence the decision to extract the teeth, or to treat the disease by conservative methods. But given the decision that conservative treatment is permissible, there is still a place for partial extraction as part of the treatment. Thus the removal of certain teeth so as to create spaces, will remove the stagnation areas and enable the remaining teeth to be kept clean without much difficulty. No rules can be laid down, for the decision as to whether the extraction of any teeth will improve the hygiene of the mouth must depend on the amount of bone destruction as evidenced by clinical examination and radiographs of the teeth.

Treatment by the Patient.

The part played by the patient is of the utmost importance. Unless there is a whole-hearted co-operation by the patient, the best efforts of the dentist will only produce a transient improvement. A careful and regular ritual of oral hygiene must be practised. Cleaning the teeth, massage of the gums, the use of silk or wool to remove debris from round the teeth, the flushing out of the mouth with a mouth-wash, are all essential. Here is the real difficulty in the treatment of pyorrhœa. So many patients mean well ; they start off with enthusiasm, which soon wanes week by week, until a rationally planned ritual degenerates into a perfunctory toilet of the mouth. Their condition lapses until the next visit to the dentist awakens a fresh spurt of enthusiasm. But each time the improvement tends to be on a lower plane, until eventually the disease reaches a stage which makes extraction inevitable. There is perhaps no disease in which it is easier to bring about some improvement ; but there are few diseases in which it is harder to maintain the improvement over any length of time.

The cry of the patient, like Naaman the Syrian, is always for the waters of the Abanah or Pharpar, which will bring some miraculous cure. But the humbler methods, based on time-honoured principles of pathology, though less showy, still remain the sheet anchor in our treatment of pyorrhœa. Used intelligently and carefully both by dentist and patient, they will not accomplish miracles, but they will arrest the progress of the disease in a considerable proportion of cases.

CHAPTER XXV.

THE TREATMENT OF THE COMMON COLD

BY

TERENCE EAST, M.D., F.R.C.P.

Physician, King's College Hospital and Woolwich Memorial Hospital.

MANY people think that there is no treatment for the common or garden cold. In fact, when the laity try to poke fun at the therapeutic insufficiencies of the medical profession, they delight in pointing out that doctors cannot even cure an ordinary cold. Of course, the very heading of this article might cause doubts to arise in a thinking mind, and the question will at once be asked: Why is the cold so common if there is any effective treatment for it at all, except hot whisky and lemon and a copious supply of pocket handkerchiefs? It may be as well to think whether we know any more about this distressing and almost universal affliction than our fathers, and whether it is really just one of the minor ailments of the human race that must be endured from time to time. As a matter of fact, a good deal of work has been done in recent years on the bacteriology of the common cold.

Anyone who has attempted to discuss what is causing the symptoms which first indicate the presence of a cold in the head will have been struck at once by the observation that just when his nose is beginning to stream, nothing may be grown from swabs taken from the mucous membrane. Years ago StClair Thomson showed that the upper nasal passages are usually sterile, but the presence of organisms would be expected in the early stages of a cold in the head. In time, that is a day or so later, it is almost always possible to grow organisms. But these vary a good deal in nature. Even *M. catarrhalis* is by no means constantly present, and may have been saddled with its name wrongly, much in the same way as *B. influenzae*. Possibly pneumococci are most often

found in the later stages, a point possibly of some importance in view of pneumonic sequelæ. The absence of organisms in the early stages of a cold in the head in many instances led to the supposition that a filter-passing virus might be the infective agent. Filtered nasal washings have been frequently injected into the noses of chimpanzees, and in a considerable proportion (about 30 to 40 per cent.) acute coryza has resulted. It seems possible that in many instances a filter-passing virus is responsible for the early stages of a cold in the head, and that a secondary infection may supervene upon the suitably prepared soil. Such a process will explain the negative bacterial findings in the early stages of some cases and the results of the filter-passing experiments. The variable findings of known organisms are explained by the secondary infections. The failure to produce infection with some filtrates might be explained by supposing that in some cases the pyogenic organisms infect primarily, as indeed the positive findings of some nasal swabbings would seem to suggest.

Vaccine Therapy.

It is unfortunate that immunity against these infections appears to be very short lived. It would be ideal to stimulate this by artificial means. Naturally, it is not possible in this instance to protect against an unidentified filter-passing virus. But many vaccines are available for inoculation against the pyogenic organism that may be associated with it. The success of these vaccines appears to be variable. Naturally a mixed vaccine should be used. In some people a good deal of protection is obtained, in others less so. Personally, the writer feels that a much more exhaustive use of these vaccines should be made than is commonly done. It is a common experience among those inoculated that a primary fairly acute coryza may occur, but never passes on to the purulent phase. By this, one supposes that protection may be obtained against the secondary infections, if not against the first phase, which may be due to a filter passer. If, as also seems possible, coryzal infections may occur primarily from known organisms, then vaccines will help to protect against these as well.

It is a matter of general experience that colds begin to become common in the autumn, about the end of September, or early in October. This is the time that the vaccine course should begin. It seems that the best results are obtained if a course of about

six injections are at this time given at weekly intervals. The dosage should be regulated so that a moderate local reaction only is produced: a general reaction should be avoided. The dose should be increased by 50 to 100 per cent. each time; the increase is regulated by the amount of local reaction. It is doubtful whether the immunity conferred lasts very long, and it is advisable to continue with further injections of the maximum dose obtained, or a little less than the maximum, throughout the winter, at intervals of about four or five weeks. Colds are not very frequent in the summer months; then the vaccine may be discontinued until the next autumn. The amount of immunity different people obtain varies a good deal, and longer intervals in the winter are satisfactory for some persons.

Local Prophylaxis.

A cold in the head usually starts with a feeling of rawness at the back of the nose in the nasopharynx, or on the upper part of the soft palate; this is usually first noticed in the morning. The first action of the infective agent appears to diminish the secretion of mucus, and the lining membranes feel dry and hot. By the end of twenty-four hours this phase is over, and mucus is freely secreted. There is much swelling of the mucous membranes, the channels are blocked, after two or three days the secretion becomes purulent; this stage lasts a variable time up to six or seven days as a rule.

Sprays.

The routine spraying of the nose by an efficient vaporizer is a very useful practice. The vehicle should be oily; liquid paraffin forms a suitable medium. The presence of the oil appears to prevent the growth of the organisms. If some substances such as menthol is added, a flow of mucus is provoked, which is the most satisfactory way of cleansing the mucosa, and thus coating it with a substance which prevents the growth of bacteria. Glegg's mixture, three parts liquid paraffin, one part white vaseline, was devised for instillation into the nose in the early stages of the cold. Poulton found that one grain of menthol to the ounce made it more effective. The earlier and the more energetically this was used in the early stages of a cold the better was the result. Such preparations may be of value in routine application as a prophylactic, and should be used every night on going

to bed. It is probable that the infection is picked up during the day, and gets a hold during the night; a morning application is then desirable. A number of preparations have been now devised—a spray such as the Pinoleum of Allen & Hanbury may be used, or thicker mixtures such as Jelopar may be squeezed in from a collapsible tube.

Once a cold has started it is difficult to check; relief is often only temporary. Probably sprays containing such substances as 1 per cent. chlorotone and 2 per cent. menthol and camphor, such as Parke, Davis & Co. produce, are as good as anything. In general, nasal douches are best avoided, as they are so easily carried into the sinuses and up the Eustachian tubes. It would be a good thing if the general public realised better the danger that may arise from a cold in the head. Every medical man is familiar with cases of pneumonia, even of the lobar type, that occur in persons who have had a cold for a day or two. The spread down the respiratory tract, to cause tracheitis and bronchitis and bronchopneumonia, is only too common. How frequently infections in the sinuses and antra, and even in the middle ear, arise from a common cold.

There can be no doubt that if people in general would take precautions such as are mentioned in this article, by inoculation and local prophylaxis, much preventable illness would be avoided. In crowded communities close contact with other persons suffering from coryzal infections is difficult to avoid. People cannot take the ideal course, which usually does much to cut short the infection, that is to say, stay in bed for two days at the beginning. But in the case of children this might be done a good deal more often than it is. With a little care and recollection sufferers might make themselves far less infectious by droplet spread than they often do. Education of the public in this respect is badly needed. It is surprising how many people think that a cold in the head is merely the result of exposure to a draught or lowered temperature only. To avoid the high incidence of this troublesome minor ailment, which may cause major consequences, may be very difficult. Immunity is too transient, and all factors are in favour of its persistence and spread. But measures on the lines suggested might do much to prevent a good deal of sickness.

CHAPTER XXVI.

THE TREATMENT OF SEASICKNESS

BY

Surgeon-Commander A. VAVASOUR ELDER, D.S.C.,
V.D., M.R.C.S., L.R.C.P., R.N.V.R. (ret.)

Surgeon to the White Star Line.

IN view of the uncertain and complex ætiology of seasickness, active treatment, combined with the observance of certain principles of general hygienic nature, can only be considered as being empiric at best. Connected therewith, certain fundamental facts must be borne in mind. The first is that the "vomiting" is only a sign or symptom, and not the disease itself. It may arise early or late in the disorder, according to individual idiosyncrasy; in some cases it never happens at all. Yet the patient is just as truly a victim of what the ancients so aptly termed "*nausea maris*," as those in whom emesis occurs. The second is that, owing to the multiple and complicated causal factors concerned in bringing this distressing condition about, no single one of the many therapeutic measures extant—not to say extinct—is a specific. What succeeds in one case almost like magic fails most hopelessly in another. It is analogous to infant feeding: there is no routine dietary scale effective in every case. It has to be altered or modified to suit the individual infant.

Turning now to active treatment. As in most other illnesses, there are two distinct forms—prophylactic and "curative," if such term be permissible in this respect. The factors of diet and drugs enter largely in both, while that of posture must be added in the latter. Prophylaxis is of what might be termed pre- and post-embarkation nature, the routine being the same in each case. The *primæ viæ* should be cleared by means of cholagogue-cathartic in the form of pil. hydrarg., gr. iv, overnight, followed by

a morning saline for two days. During the daytime pot. brom. is given in ten-grain doses, thrice daily, after food. The diet should be plain and simple, avoiding anything "rich" or "bilious" in nature. Alcohol need not necessarily be abstained from, except perhaps to excess in the form of too many farewell parties prior to embarkation. The latter are not infrequently the cause of many early cases of "seasickness"—more strictly definable as the vomiting of alcoholic gastritis. By following the régime outlined above, preferably for two or three days prior to and after sailing, omitting of course excessive purgation, the majority should escape or minimise the pangs of seasickness while acquiring their "sea-legs."

As to "curative" measures, much can be attained by them when the right combination for the individual patient has been found. Unfortunately, like lunacy, seasickness seems to imply a social stigma, which in turn prompts many to hide it, suffering more or less silently in the privacy of the stateroom instead of calling in the ship's surgeon early. When, after prolonged use of patent preparations which have failed to do anything but half poison them, and driven desperate by suffering, they do call in the surgeon, then his task is at once increased by the debilitated and partly drugged condition of the patient. He cannot perform the miracle not infrequently expected of him in such circumstances.

As already stated, therapy is summed up in posture, diet and drugs. The prone position, preferably on deck in the fresh air and well wrapped up against cold, with or without hot-water bottles, as indicated, and as near the centre of the vessel as possible (to diminish the effect of pitching, usually most active in bringing this condition about), is undoubtedly the best. Failing this, usually for reasons of diffidence about appearing in this state in public, the patient should remain in bed and the cabin be kept ventilated to the utmost. A tight abdominal binder—bath towel kept in position by safety pins—adds to physical comfort.

On the subject of diet, there are three very popular fallacies commonly held by the travelling public. One is that of forced feeding; the other consists of starvation; while the third lies in abstention from all liquid. In the main, these are all wrong, and cause unnecessary suffering, besides being quite ineffective in preventing or diminishing seasickness.

Normally, in between 75 to 80 per cent. of cases, all thought, sight or suggestion of food is repellent to the patient; in many

instances it will arouse a shudder of disgust. Of the remainder, some will take food only for the sake of eating—to avoid getting “weak,” as they usually put it—while the rest eat because they feel like it, frequently expressing desire for some special item they seem to fancy, somewhat akin to the peculiar “longings of pregnancy” experienced by some women when in this state. Hence, in my practice I never force food, while allowing any article the patient may fancy, no matter how strange it may seem. Provided it is really fancied, there is more chance of its retention by the stomach than the mildest of pre-digested foods.

Where liquids are concerned, however, it must be remembered that prolonged vomiting leads to a state of acidosis and dehydration, two conditions studiously to be avoided. The best method is to give repeated draughts of water containing sod. bic. in the proportion of gr. v to the ounce. Apart from counteracting these undesirable factors, the water gives the patient something to be sick with. It diminishes the pain and discomfort when the desire to vomit supervenes, instead of being racked by dry retching because of having nothing to reject. If the patient prefers other fluids, and will take them voluntarily in between attacks, then I only insist upon half a tumbler being drunk after each spell. Ice to suck is refreshing, besides ensuring an intake of water in the process.

Referring to alcohol: here, again, the very thought of it will nauseate some patients, while others often express a wish for it on their own account, and not because of its vaunted virtues. On the basis of fancy, I defer to the patient's wishes; and in the majority of cases with success. While on this subject, champagne, plain or in combination with stout, has been much extolled. Here again the factor of idiosyncrasy enters. However, in my experience at sea of many years, I have never seen champagne succeed where a good dry ginger ale failed. But the latter must be dry. Presumably it is the carbonic-acid gas content in both which relieves the patient, and not the alcohol. Also, where no food whatever is being taken, and the stomach is more or less empty, fizzy drinks may cause unpleasant distension. Therefore before administration they should be stirred up to remove excess of gas. A plain, dry biscuit—water cracker—should be munched at the same time. If acceptable to the patient, the ginger ale can be “stiffened” with brandy, gin or whisky, according to taste. But to repeat, fluid of some sort is essential in all cases, and water tastes less nasty

on its return than other liquids, besides acting as a form of gastric lavage in readiness for the day of recovery—the day when in all probability food will be eaten to excess to make up for lost time and opportunity, in so far as the average liner's menu is concerned.

Drugs in the treatment of seasickness may be considered in three separate and distinct categories, as follows :

(1) "Nostrums," the effect of which is comparable to "old wives' tales," depending entirely upon the psychological reaction or the recipient, *e.g.*, "seasick plasters"; a mercury bag worn round the neck, ear protectors, etc.

(2) Proprietary preparations, which, if not absolute rank frauds, contain one or more of the orthodox ingredients in varying combinations and proportions ordinarily used for this condition. They are sold at enhanced prices to pay for extensive advertising, and help the Inland Revenue sale of Patent Medicine Tax stamps. In some cases they are efficacious; while total failures in others. Apart from the high cost to the public, the main objection to them lies in their content: in many cases of highly toxic drugs of depressant nature, the unregulated absorption of which constitutes a distinct danger. This is especially so in cases of persons unduly susceptible to them. I have seen many cases of typical atropine and hyoscine poisoning, to say nothing of overdosing with veronal, chloral and bromides, resulting from continuous ingestion by patients in the vain hope of obtaining relief. Incidentally, that seemingly universal panacea—*aspirin*—is quite useless for the headache accompanying seasickness; in bulk and of doubtful purity, it may also set up an acute gastritis as an aftermath.

(3) Orthodox, if such term can be applied to what is little less than empiricism. In this connection I would here register a strong protest against shore practitioners sending patients to sea armed with pill, potion or powder—the contents of which are unknown to the latter—and with instructions to take until relieved. It can be accepted as axiomatic that if six doses fail to relieve, then the mixture should be changed. It is futile to persevere with an obvious failure, quite apart from the physio-pharmacological injury which may be inflicted upon the patient thereby. In other words, family physicians should not attempt "absent treatment" upon their patients afloat, but refer them to the man placed on board for this purpose—the ship's surgeon—and advise them to consult him prophylactically rather than curatively.

Having attended to the factors of diet, posture, and the alimentary canal, my routine is as follows: a hypodermic injection of

Strychnine sulph. gr. 1/32

Atropine sulph. gr. 1/120

Aq. distill. min. 5

Three injections are given at four-hourly intervals. If unsuccessful as to relief or actual cure, other measures are then tried. Three injections suffice as a rule, but in the case of "bad sailors" more may be required according to local weather conditions.

The objections to this method are two-fold: Many patients are needle-shy, and refuse to submit; to others, mention of a hypodermic at once suggests "dope." After assurance to the contrary on this point, and submitting to treatment, many patients, on waking up from the restful sleep usually accompanying it, will accuse the practitioner of bad faith, and refuse to continue. Its advantages are the high percentage of efficacy, also that given in this fashion, the drugs are allowed full scope, as they cannot be rejected.

In addition to the relief of simple seasickness, there is also the co-existence of pathological conditions to be considered wherein unrelieved or violent vomiting may be distinctly dangerous. Atheroma, with high blood-pressure and liability to hæmorrhage; herniæ of various nature, and possible strangulation; breaking down of recent operation scars; pulmonary tuberculosis, which should never be sent to sea except unavoidably; early pregnancy, up to third or fourth month, after which mishap is not to be feared as a general rule. Incidentally, pregnancy between the fourth and eighth month is no contraindication to a sea voyage. Before or after these dates excessive emesis may result in a miscarriage or premature labour.

In cases of atheroma with palpable high pressure—as a rule patients in the throes of seasickness do not willingly submit to sphygmomanometer readings—liq. trinitrini, in minim doses under the tongue every three or four hours, is strongly indicated. The vomiting, if not actually relieved, is much improved and the tension reduced. The best method is by using an old-fashioned hypodermic syringe, graduated to deliver minim doses by means of a movable screw collar on the stem of the plunger. Or, alternatively, by a pipette fitted with a special nipple. The ordinary nipple is too soft, and owing to movement or vibration of the ship may cause an overdose. The special nipple is made from two

inches of stout rubber tubing. One end is closed by a suitable-sized cork covered with sealing wax, and the other applied to the pipette in the usual way. A draught of water is given to take away the burning sensation of the drug. In the event of overdose, a hypodermic of strychnine, gr. 1/32, and spts. ammon. aromat. to the nares, acts as antidote.

The official tab. trinitrini are slower in action, and may be rejected, whereas the liquor is quickly absorbed under the tongue, and its effect noticed within about twenty seconds, especially in regard to the headache so frequently present in these cases.

Apart from the above, the bromides, chloral hydrate, chlorotone, valerianates, singly or in combination, may be tried. They should be given in full doses until relief or saturation of the patient is reached. A useful mixture is as follows :

Pot. brom.	gr. 10
Spt. amm. arom.	dram 1/2
Aq. chlorof.	ad oz. 1

This is given hourly for six doses, the dose being repeated if not retained for half an hour.

For patients in bed, rectal suppositories of choral hyd. and pot. brom., $\bar{a}\bar{a}$ gr. 10, made with cocoa butter, or theobroma, can be used. The rectum should be previously cleared by common enema, otherwise the suppository may act mechanically as such.

Mention by name of all patent preparations and anti-nausea specifics has been purposely omitted. Some help some sufferers, but no single one helps all. Moreover, as already stated, they should not be taken indiscriminately or without medical supervision.

In conclusion, prospective sea travellers should be encouraged to consult the ship's surgeon early, instead of suffering unduly before so doing. Also certain cabin attendants are met with who for personal reasons of their own discourage the attendance of the surgeon for such a minor ailment as *mal de mer*. Seasickness is a definite disorder, and not a social slur. Therefore the victim is entitled to, and should receive, the best that medical knowledge can provide, and should not be allowed to suffer unnecessarily.

For the readers interested in this subject, fuller details will be found in "The Ship-Surgeon's Handbook" (Baillière, Tindall & Cox); "Notes on Seasickness," *Journal of the Royal Naval Medical Service*, July, 1931; and *The Medical Press and Circular* of May 23rd, 1928, and October 10th, 1928.

CHAPTER XXVII.

INSOMNIA AND ITS TREATMENT

BY

E. B. STRAUSS, M.A., D.M., M.R.C.P.

Assistant Physician to the Cassel Hospital for Functional Nervous Disorders.

ALTHOUGH this chapter is intended to be severely practical rather than academic (so much so that the reader will seek in vain for a bibliography, which is absent for the very good reason that the writer has not relied on other works for the composition of this paper), certain theoretical questions must first be briefly touched upon if what follows is to be intelligible.

The Nature of Sleep.

The work of Pavlov and his school has, I think, given us the best and truest conception of sleep in defining it as the *general inhibition of cortical functions*.^{*} It has the advantage of (a) being experimentally verifiable; (b) squaring with all the pre-existing theories which have not been demonstrated as false; and (c) being able to dispense with these other theories nevertheless. Just a few words about three of these rival theories: (1) Sleep is due to the accumulation of specific fatigue toxins. This theory has so little to support it that it can be abandoned without further discussion. In fact, if one desired to define sleep in teleological-biological terms, one might say that *sleep is a biological function concerned with the prevention of exhaustion states*. (2) Sleep has a physico-chemical basis. This is certainly true, but it in no way interferes with the conception of sleep as inhibition. Surely, cortical neurones when exercising inhibitory functions must be regarded as being in a different physico-chemical state than when aiding in excitatory functions. It is perfectly conceivable and

* Those who are unfamiliar with the concept of physiological inhibition and Pavlov's theory of sleep will find a summary in Chaps. 14 and 15 of "Recent Advances in Neurology," Brain and Strauss, London, 1930.

consonant with empirical facts that neurones can be put into such a state directly by physico-chemical agents as well as by an irradiating wave of inhibition of reflex origin. (3) Sleep is the action of a specific sleep centre. There is a certain amount of impressive evidence in favour of the existence of a sleep centre situated in the neighbourhood of the hypothalamus. Even if that be conceded, the action of that centre may either be to initiate and irradiate inhibitory impulses, or, by its influence on neighbouring metabolism-controlling centres, to produce the physico-chemical conditions favouring general cortical inhibition. We can then assume that sleep is identical with general cortical inhibition, and it follows as a logical corollary that *anything which interferes with the spread of cortical inhibition can produce sleeplessness.*

Partial Sleep.

There are all grades of sleep, ranging from somnolence, in which conscious faculties *seem* to be intact, the hypnoidal state, hypnosis and "normal" sleep, to deep narcosis and coma. All these states may be regarded as different degrees of inhibition. Thus there is no essential difference between "normal" and hypnotic sleep. In hypnotic sleep the only difference is that all cortical neurone-groups are inhibited, with the exception of those maintaining perceptual contact with the hypnotiser. But it is a well-known fact how "selective" even normal sleep is. For instance, during the War I could sleep through the heaviest bombardment, whereas my name whispered by an orderly could awaken me instantly. I mention these forms of partial sleep because it is important to be able to reassure a patient that all forms and grades of sleep, however produced, are beneficial. To be personal again: during the War I have been so tired that I have slept on the march (*i.e.*, been able to carry on the motor actions of walking in a state of somnolence), and have felt all the better for it. Further, lectures always produce in me a delicious state of somnolence, from which I emerge much refreshed.

The Effects of Sleeplessness.

Experiments which have been conducted up to date to ascertain the effects of sleeplessness on man all show that the results of absence of sleep alone are almost negligible. The undesirable effects, such as they are, are the result of the motor restlessness which is almost inseparable from periods of insomnia, and all these effects

disappear after twelve to fifteen hours' sleep, even if the period of insomnia has lasted for a hundred hours or more. The experiments also showed that subjects who were absolutely certain in their own minds that they had kept awake the whole time, were observed often to fall into a state of somnolence. This adds weight to our words when we assure patients who claim to have passed several absolutely sleepless nights that they have almost certainly had several hours of somnolence.

There is no evidence to support the view that insomnia can cause insanity. The idea that insomnia was in itself a cause of insanity arose from a misinterpretation of the fact that in many forms of psychosis insomnia is a constant feature, and may be the initial symptom.

Ætiological Survey of Insomnia.

As we have already said, anything which interferes with the normal physiological spread of cortical inhibition may lead to insomnia. Such factors may be either physical or psychic.

(a) PHYSICAL FACTORS.

(1) *Toxic and Chemical Factors.*—As is well known, insomnia may complicate the symptomatology of almost any toxic illness or morbid condition in which metabolism is disordered. This is especially true of typhus, meningococcal meningitis, rickets, acute epidemic encephalitis, influenza and pneumonia. The insomnia which is so commonly associated with arteriosclerosis is often of dual origin. The deficient elimination of toxins by the kidneys probably leads to insomnia of toxic origin; but often the most serious feature of arteriosclerosis is the accompanying arteriosclerosis phobia which leads to hypochondriacal anxiety pre-occupations, and hence to insomnia of psychic origin. These phobias are very frequently produced by tactlessness on the part of the doctor; and once established, they can often be removed by persuasion and suggestion.

The chief chemical factors concerned with the production of insomnia are, of course, tea, coffee and tobacco. There are, undoubtedly, persons who show an idiosyncrasy either for caffeine or the alkaloids and other bodies in tobacco smoke. It may not be enough to forbid tea or coffee merely the last thing at night, as in some patients these drugs, even in minute quantities, have a very protracted effect. In such cases it is best to forbid tea and

coffee (or smoking) altogether. The insomnia associated with true exhaustion states, especially when resulting from motor restlessness (*e.g.*, the incessant hyperkinesis of chorea gravis) is probably toxic in origin.

(2) *Pain, Discomfort, Dyspnoea, Cough, Uncomfortable Temperature and the like.*—All these factors may clearly cause sleeplessness. The obvious way to treat such insomnia is to remove, so far as possible, the painful or irritant stimuli. But the cause is not always apparent to the patient. Many persons are either too hot or too cold in bed without knowing it; and some patients are not fully aware that their beds are uncomfortable. One patient consulted me for insomnia, complaining that he would not mind the insomnia so much if he did not feel so cold all night. It did not occur to him that his insomnia might be due to his being cold. I suggested a hot-water bottle, but he demurred on the grounds that it was “unmanly.” I asked him how he slept, and it transpired that he slept under an open window with his arms outside the bed. At first he said that he did not know why he slept in that way, but after a little persuasion it emerged that it depended on a masturbation phobia. As a child he had been made to sleep with his arms outside the clothes to prevent “bad habits,” and the idea of cold air all night was associated in his mind with cold baths, which are frequently prescribed as a charm against the same habits. His insomnia disappeared when I persuaded him to introduce a little element of “fug” into his sleeping hours. Of course, the reassurance he received on the matter of masturbation may also have helped.

(3) *Food Habits.*—Some people whose gastric content is slowly voided take too large meals too soon before going to bed, and are in consequence kept awake by the barely perceptible gastric discomfort. Others, who have been told that a loaded stomach at night gives rise to insomnia, go to the opposite extreme and eat too little at night. They complain that they wake in the early hours of the morning and cannot get to sleep again. What has really awakened them is hunger. If they drink a glass of milk and eat a biscuit when they wake up they will soon learn to fall asleep again.

(b) PSYCHIC FACTORS.

Clearly, excessively painful, frightening or generally exciting emotions are likely to check the irradiation of cortical inhibition

which we call sleep. Nor is it necessary that the emotions be appreciated as such at the level of consciousness; for "unconscious" psychic conflicts can produce the same results.

(1) *Insomnia in the Psychoses.*—It is probably the over-active affective stimulation which is responsible for the insomnia met with in the psychoses (especially manic-depressive psychosis and schizophrenia). Since it is impossible at present to change the affective pattern in these states by psychotherapy, the insomnia must be treated symptomatically by drugs, hydrotherapy and the like.

(2) *Insomnia Complicating other Functional Nervous Disorders.*—Insomnia is often a feature of the various kinds of hysteria, anxiety states, obsessional-compulsion neurosis, and other psychoneurotic reactions met with in general and specialist practice, even when it is not one of the symptoms of which the patient chiefly complains. Quite 50 per cent. of the patients undergoing a systematic course of psychotherapy require hypnotic drugs at one time or another. In these cases the insomnia is, of course, due to the painful *affect* associated both with their illness and with the treatment. In the course of psychotherapeutic treatment, whether by persuasion and explanation or by deep mental analysis, patients are made to face up to painful situations or distasteful factors in their own make-up which have been previously relegated to the periphery of consciousness or to the depths of the psyche. It is my practice to treat such insomnia symptomatically with drugs. Persistent bad nights complicate treatment unnecessarily, and the insomnia always disappears when the general neurotic state has been mastered.

(3) "*Worry.*"—"Real" worries, by which I mean worries from outside—business difficulties, troublesome love affairs, domestic strife and the like—can "play upon a person's mind" so as to disturb sleep. If the family doctor would be a sympathetic and helpful audience and confidant for about half an hour, instead of reaching out for his prescription book the moment sleeplessness is mentioned, he would be able to avoid having many chronic insomniacs amongst his patients. A short holiday often succeeds in putting an end to that type of insomnia. But the physician must know all the facts of the case. It is no good sending a man away for a week's holiday with his wife if the marital situation is really responsible for the man's worries.

(4) *Insomnia as a "Conversion" Symptom.*—I am using the term "conversion" in a somewhat wider sense than the strictly

psycho-analytical (Freudian). By insomnia as a conversion symptom, I mean insomnia which is produced to serve the patient an unconscious purpose—either to gratify some desire which he is unable to admit into full consciousness, to mask some repressed fear or other, to serve as self-punishment for some unconscious “crime,” and so on. In these cases insomnia may be the chief or sole symptom complained of. As a rule it only responds to analytical psychotherapy. As an example of an unusually simple case of this nature: It became quite clear in the course of analysis that one patient had developed insomnia as a means of providing an excuse for sleeping apart from his wife, for whom he had an unconscious (*i.e.*, unadmitted) aversion. In his (and his wife’s) ideology, sleeping in separate rooms would not have been respectable, in fact almost immoral. He was able to persuade himself that the slightest noise, such as his wife’s turning over in bed, kept him awake or woke him up. Further, the symptom seemed to express literally (with the help of a *double entendre*) the sentiment: “I cannot sleep with my wife.” Even after his symptom had achieved its object (*i.e.*, procured him a separate bedroom) it persisted as a punishment for his unconscious “crime.” The appreciation by the patient of the (previously unconscious) psychological causal nexus effected a cure.

(5) “*Agrupniaphobia*.”—I have coined the word *agrupniaphobia* to denote a morbid anxiety concerning insomnia. It is perhaps the commonest cause of a *persistent* insomnia when sleeplessness is the sole symptom complained of, and can give rise to the most distressing symptoms: feelings of exhaustion, loss of weight, and the whole gamut of “neurasthenic” symptoms. It can, in addition, complicate insomnia arising from any other cause. There are many analogies here between the results of *agrupniaphobia* and masturbation. [The evil effects of masturbation result not from self-abuse as a physical act, but arise from the psychic dragon’s teeth that it sows: feelings of being a unique “criminal,” a pervert or what not, the conviction that the habit will drive him mad, destroy his will power, render him impotent, and so on—ideas of references (*e.g.*, that people can detect this “beastliness” in his face); this last leads on to blushing and erythrophobia.]

A person has a succession of bad nights for some cause or other, and consults his doctor. The doctor looks grave, talks about

insomnia (to patient's mind) as though it were a disease in itself, and prescribes a sleeping draught. Possibly the dose prescribed is not nearly strong enough, and it fails to act. He now has the impression that his must be a very grave case of insomnia indeed ; and so the vicious circle starts.

It is usually perfectly easy to persuade people that what is keeping them awake is a fear of insomnia and its "inevitable consequences."

Another phobia which frequently accompanies agrypniaphobia is a phobia of hypnotic drugs. This makes it appear to such patients impossible that they can ever get proper sleep again. Even if they are persuaded to take a sleeping draught occasionally, the worrying thought that they are becoming incurable drug addicts as often as not prevents the draught from taking effect. Only too frequently it is the doctor himself who, all unwittingly, implants this phobia in the patient's mind by his manner when prescribing the sleeping draught.

It is hard for a physician to grasp to what an extent he is taken seriously by the majority of his patients. The doctor is not 100 per cent. right, but 200 per cent. A grunt from the doctor, a shake of the head, the slightest word, express volumes. Consequently, the power of suggestion exercised by the doctor for good or evil is enormous. A very appreciable percentage of the patients suffering from psychoneuroses are suffering from doctor-created illnesses.

(6) *Insomnia in Children*.—Psychogenic insomnia in children is by no means uncommon. As often as not it is an expression of normal childish "negativism," a reluctance to go to bed or get to sleep before the grown-ups. It is usually easy to reduce cerebral activity in children (without their noticing it) an hour or so before going to bed, by playing increasingly quiet games with them, or reading aloud not too exciting stories ; and going to bed itself can be made interesting and pleasant by introducing little games and surprises into the ritual of undressing and "bed-time" in general (Gillespie). Insomnia in children may also be due to a fear of nightmares or an enuresis phobia. Sometimes the child regards the nightmares as so dreadful that he avoids all reference to them in speech, and will even deny that he has them. Both these fears require treatment by psychotherapy. Insomnia in children may also be a conversion symptom (in the sense described

above). The "purposes" served may be a means of compelling parental attention and sympathy, or a means of revenge (*i.e.*, getting even with the mother or nurse who are always prohibiting what is agreeable).

Children with a fear of the dark—sometimes a cause of insomnia—should be allowed nightlights.

Treatment of Insomnia.

I have already given many implicit and explicit indications as to treatment. To supplement these—the cardinal principle of all medical treatment, of course, holds good: (a) Diagnose the cause of the symptoms complained of; (b) as far as possible treat the cause of the symptom rather than the symptom itself. Insomnia is a symptom, not a disease *sui generis*; I have yet to be convinced of the existence of an *essential insomnia*.

Make a thorough physical examination in all cases. Insomnia may be the initial symptom of some organic disorder, and in any case, the patient usually requires reassuring that there is nothing radically wrong with him. Take a very careful history, inquiring into all possible sources of external and internal worry and conflict, habits, domestic and business situation, and so on. A full history combined with a careful physical examination should make it perfectly easy to assign the sleeplessness to one of the categories listed above.

Insomnia of physical origin must, of course, receive treatment calculated to abolish or relieve the physical cause. In many cases of this kind the use of hypnotic drugs will be indicated in addition.

The treatment of the restlessness and insomnia associated with many of the psychoses belongs to psychiatry proper, and is outside the scope of a general article such as this. The only psychoses which the general physician is likely to be called upon to treat for any length of time are the milder manic-depressive attacks and the mild pre-senile and senile dementias of arteriopathic origin. According to my experience, the most useful hypnotic in depressive attacks is Medinal (gr. 10). I believe it to be perfectly safe to continue the drug over long periods provided that a careful watch is kept for signs of poisoning (a clinical rarity). The symptoms of poisoning by the barbitone group are cerebellar, especially a staggering gait; sometimes erythematous rashes appear. In insomnia associated with hypomania, ammonium bromide (gr. 10 t.i.d.),

and paraldehyde at night (if necessary up to drams 3 or drams 3½) are indicated. The most suitable hypnotic drugs for arteriopaths are the bromides (ammon. brom., gr. 20-40), chloral (gr. 10-20), and paraldehyde. Elderly arteriopaths who sleep badly often respond well to a glass of whisky or hot grog last thing at night.

Insomnia due to worry and intellectual and emotional strain responds well to general and common-sense measures, combined with psychotherapy in the form of reassurance and persuasion from the doctor. The measures include relaxation exercises, massage, a "nap" during the day (the patient must be told that sleeping in the daytime in no way prejudices his chances of a good night's rest—on the contrary), exercise in the open air (the patient must realise that this is undertaken *not* to produce fatigue or exhaustion, but as a general measure). The type of therapeutic conversation indicated will be described in the paragraphs dealing with the treatment of aggrupniaphobia. It is as well to supplement this kind of treatment during the first week with hypnotic drugs. I suggest Medinal in cachets, gr. 10, the first two nights, gr. 7½ the following three nights, and gr. 5 the last two nights.

Psychogenic insomnia which arises in connection with other forms of neurosis, or which is suspected as being a conversion-symptom, requires skilled psychotherapy.

Insomnia in children will nearly always respond to general and common-sense measures. In stubborn cases, hypnotic drugs may be given, the safest being ammonium bromide and chloral, in doses according to the child's age.

Treatment of Aggrupniaphobia.

By far the majority of cases of chronic insomnia met with in general practice are due to this cause, and the general physician can obtain the most brilliant results by psychotherapy without the continued use of drugs. The following is a résumé of the type of therapeutic conversation I might have with such a patient :

"I have examined you carefully, and I can assure you that you are fundamentally sound in wind and limb. Sleep is a habit—sleeping well is a good habit ; sleeping badly a bad and stupid one. You have told me all your story, and that has convinced me that you are not suffering from a disease called insomnia : in fact, there is no such disease. You have acquired a bad habit merely. The reason why you cannot sleep is because you are afraid of not

being able to sleep, and afraid of the consequences of not being able to sleep. There is no need to be afraid of not sleeping.

"Experiments have shown that one can get on well with next to no sleep. When I was in Venice one September, I found that the hot and sticky climate, combined with an equal dislike on my part of mosquitoes and mosquito-netting, made sleep almost impossible for me. Instead of going to bed I used to roam the streets nearly all night, and have a full day's sight-seeing and bathing during the day-time. For a fortnight I suppose I did not sleep more than four hours in the twenty-four, and I felt thoroughly fit the whole time.* You do yourself no harm by lying awake all night, provided that you do not worry about not getting to sleep and do not toss about and get "hot and bothered." It is restlessness and worrying which has pulled you down, not the lack of sleep in itself. From now onwards go to bed at night not caring whether you sleep or do not. If you sleep, so much the better; if you do not, and if you lie quite still, with all your muscles relaxed, it will do you almost as much good. If you want a dodge to assist sleep, here is one: Have your watch by your bed and listen intently to its ticking. After a time the ticking will seem to become fainter. You will soon fall asleep after that, provided that you concentrate on the watch and not on the idea of sleep. This is not 'magic'; it is a scientific fact that any monotonously repeated gentle sound favours the onset of sleep."

Drugs: How to Administer them and What to Administer.

This is not the place for an article on the pharmacology of sleep. I wish only to conclude with a few hints on how to administer hypnotic drugs, together with a few remarks on the drugs of which I myself have any considerable experience, and no others.

How.—(1) When prescribing hypnotics, do not give the patient the impression that you are half-afraid of the step you are taking. (2) If possible, exhibit the drug in cachet form. Patients then need not know what drug they are taking (especially if the physician does his own dispensing), and come to imagine that that particular drug is the only one that suits them. Further, the dose can be reduced at will, unknown to the patient. (3) Do not delay the use of hypnotic drugs once you have decided that they are indicated. (4) Do not be afraid of giving *full doses* over a

* When one can, it is always good to relate personal anecdotes—true ones if possible.

short period. (5) Be prepared to meet a possible drug-phobia on the part of the patient with reassurance and persuasion. (6) If drugs are required over a long period, ring several changes on them.

WHAT.—*The Barbitone Group*.—In my experience the best all-round “general purposes” hypnotic is Medinal (mono-sodium barbitone). None of the other innumerable barbitone preparations appears to me to have any special advantages over Medinal (gr. 5–10).*

“*Allonal*” (tab. 1–2) is an excellent hypnotic when pain or discomfort (which is not sufficiently severe to require opium derivatives) is present.

“*Somnacetin*” is indicated instead of Medinal in arteriosclerotic cases.

“*Chloral-hydrate*” (gr. 5–30, always in solution) has impressed me as being an excellent and safe drug in spite of all the “bad marks” chalked up against it in the text-books. I use it chiefly as one of the changes to be rung on Medinal.

“*Sulphonal*” (gr. 10–30) is another useful drug to alternate with Medinal. It has a very slow action. I always give Sulphonal in cachet form at 6 p.m. It should not be given for more than a week without a break.

“*Paraldehyde*” (dram 1–3) is an excellent and very rapid hypnotic. It is especially useful in excitable and restless patients, and in alcoholics. It should not be given for more than a week on end, as, in spite of its filthy taste and odour, Paraldehyde habituation does occur.

“*Adalin*” (gr. 7–15) is a weak hypnotic which acts fairly rapidly. It is useful in mild insomnia due to worry or excitement, and can be safely used over long periods.

“*Amylene Hydrate*” (min. 30–80) may be alternated with Chloral and Medinal with advantage.

Potassium, Sodium and Ammonium Bromide.—Generally speaking, the bromides are poor hypnotics when used alone. But they are of great value when prescribed (in about 25-grain doses in the case of adults) along with other hypnotic or analgesic drugs to supplement the effect of the latter.

In spite of the literally enormous number of hypnotic drugs, both “official” and proprietary, which are offered to one’s choice, it is my opinion that the few very ordinary ones mentioned above will be found to cover almost all one’s requirements.

* The doses given here are not necessarily the official doses.

CHAPTER XXVIII.

OTITIS MEDIA IN CHILDREN

BY

E. WATSON-WILLIAMS, M.C., Ch.M., F.R.C.S.

Surgeon-in-Charge, Ear, Nose and Throat Department,
Bristol Royal Infirmary.

Ætiology.

SUPPURATIVE otitis media* is a common condition, and may be seen at any age, but the first attack is nearly always in early life. It occurs in two-thirds of all cases between the years of three and fourteen, when the exanthemata take their toll, but even in this period the "common cold" is the most frequent factor determining an attack. The incidence is as high during the first year of life as in later childhood, and actually highest during the second, and here one of the most frequently alleged causes is teething. Teething, we all know, is a simple, painless, physiological process, but many children at this tender age are ignorant of this, and in addition to gastro-enteric or bronchial disturbances we often encounter the story: "the ears ran with every tooth he cut."

Just as an acute naso-pharyngeal coryza, from whatever cause, precedes almost every attack of otitis media, a chronic infective condition in this region both predisposes to attacks and hinders resolution. We have not yet begun to appreciate that chronic sinusitis is as common in children as in adults. But the commonest manifestation of disease here is adenoid hypertrophy. What is the cause of this? In many cases no doubt repeated infection from parents or associates suffering from tonsil or sinus infection may be looked upon as the reason for the child's adenoids. In a small but not negligible number congenital syphilis is responsible. But in the majority no such explanations can be found, and the answer must be sought in unfavourable general hygienic conditions, especially unsuitable (highly farinaceous) diet. The prophylactic

* Throughout this paper, otitis media refers to the suppurative form.

value of fresh air, suitable clothing and cod-liver oil are well established (though not always utilised), but few children of a year old get the meat and fruit to which their teeth entitle them, and many get none at all.

Prophylaxis.

These considerations merit serious attention, for the prophylaxis of otitis media in the young, and therefore of the great bulk of adult deafness, lies in the hands of the family doctor. Once

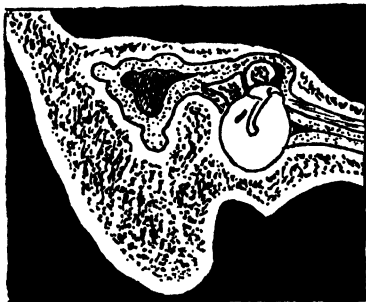


FIG. 1.—Vertical section in the (very oblique) plane of the tympanum. Cancellous or infantile type of mastoid. At birth there is no mastoid process; at one year old it has developed. The antrum is relatively large, there are no cells, the bulk of the process is composed of cancellous bone, and the cortex moderately thick. This type persists in about five per cent. of adults.

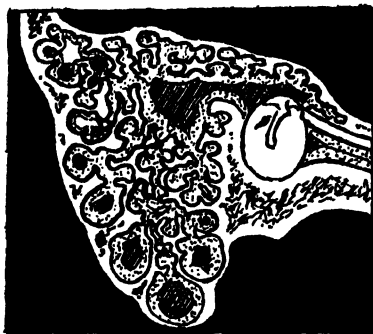


FIG. 2.—Similar section of cellular mastoid (75 per cent. of adults). The incus and head of malleus removed. Cell development begins in the first year, and is nearly complete by the tenth. The process is convex within and without, the cortex thin, cancellous bone almost absent, and the bulk of the process made up of a complex labyrinth of cells with thin walls, the peripheral cells tending to be larger than the central.

adenoid hypertrophy or sinusitis is established, the only effective treatment is surgical, either to cut short otitis media or to prevent recurrent attacks; irreparable harm may be done to the hearing by procrastination. Tonsillectomy by itself has little influence on otitis media.

Course.

Once the ear has become infected the progress of the case depends on three factors:

(a) Natural drainage *via* the Eustachian tube; the importance of naso-pharyngeal disease is seen here, for "adenoids" do not

FIGS. 1-5.—Anatomy of types of mastoid process, semi-diagrammatic; the position of the tympanic membrane is indicated. (The Eustachian tube is drawn too wide.) Mucous membrane represented as if swollen by inflammation in fine dotted stipple; discharge lying in the cells in heavy diagonal hatching.

cause mechanical obstruction to the tube, but their presence leads to congestion of the mucosa and narrowing of the lumen.

(b) *Anatomical Configuration of the Middle Ear.*—Drainage that would suffice for the small area of mucosa of the process shown in Fig. 1 might be inadequate for the exudation from the much larger area of Fig. 2; the vascular bone of the former might be more readily invaded than the dense bone of Fig. 3.

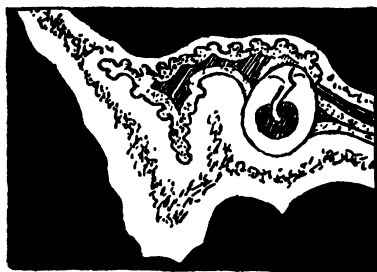


FIG. 3.—Similar section of sclerotic mastoid (20 per cent. of adults). The process is flat or concave, external surface deeply grooved, dura often dips down over the antrum. Antrum small, cells very poorly developed, cortex very dense, cancellous bone persists to a small extent. This type of mastoid is seldom seen when operating for "acute mastoiditis," but forms the bulk of those met with when carrying out radical operation for chronic disease.

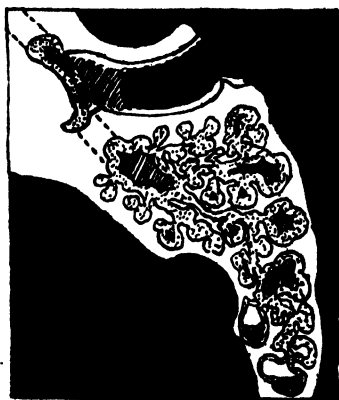


FIG. 4.—Nearly horizontal section of same bone as Fig. 2. Showing the great obliquity of the middle ear tract and tympanic membrane; and that cells may extend back well behind the groove of the sigmoid sinus. Position of aditus and Eustachian tube dotted.

(c) *The Virulence of the Infection and the Patient's Powers of Resistance.*—From this point of view we can distinguish clinically several varieties of otitis media.

Types of Disease.

(1) *The Toxic.*—Fortunately very rare. From the outset the child is obviously very ill, listless, drowsy, perhaps delirious, with dry, red tongue, parched lips, sunken facies, thready pulse; the temperature is not much raised, ear pain is moderate only, the discharge is thin and sanious, and if the mastoid is opened it is neither engorged nor full of pus. Opinion differs as to whether one should operate early in these cases; my own opinion is in favour of this course, but whatever is done the mortality is high.

(2) *The Hæmolytic.*—Associated usually with influenza. The pain, tenderness and deafness are severe, the temperature high;

the tympanic membrane shows purple hæmorrhagic bullæ before rupture, the initial discharge is bloody and profuse, and the mastoid process, if opened early, shows little pus in the cells, but the bone runs blood. Early incision of the membrane and early operation on the mastoid are necessary here for the sake of the hearing.

(3) *The Histolytic*.—This type is specially prone to occur in measles and scarlatina. Pain is often of short duration, and the membrane ruptures early, a large perforation being produced with free purulent discharge; if the mastoid is opened the intercellular trabeculæ may be found to have disappeared within a very few days of the start, leaving the whole process nothing but a shell. Extension of suppuration to surrounding structures is specially liable to occur.



FIG. 5.—Vertical section of same bone at right angles to plane of tympanum, to show (a) head of malleus, with ligaments, tendon and swollen mucosa almost obliterates the cavity of the attic; (b) swollen mucosa over promontory is virtually in contact with margins of any central perforation.

(4) *The Purulent*.—Most common type. Pain may be of moderate severity and often intermittent; the temperature is $101-103^{\circ}\text{F.}$; the membrane is injected and may bulge, perhaps for two or three days before rupture occurs, and when this happens the perforation is small; the discharge is free, and at first purulent, and other symptoms subside when

it occurs; if the mastoid is opened the cells are full of pus, but the bone is often not grossly diseased.

(4a) *The Pneumococcal*.—Signs and symptoms are relatively mild, but the infection tends to persist; it is said that all local signs may appear to clear up, but there is always *some* degree of deafness and tenderness in an unresolved case, even when the membrane has healed, with occasional fever; this variety tends to smoulder quietly until some complication (extra-dural abscess or sinus thrombosis are the commonest) occurs with little warning.

(5) *The Mucous*.—A milder type of (4). The perforation may be a mere slit; the discharge is mucoid almost from the start, and often thick; if the mastoid is opened, the cells are filled with swollen mucous membrane, with little discharge.

(6) *The Benign*.—With only slight pain and fever, the membrane ruptures; the discharge rapidly ceases, and healing may occur within a day or two.

Signs and Symptoms.

From what has been said it is clear that the signs and symptoms vary considerably in degree. *Pain* and *fever* are generally definite, and indicate the nature of the trouble. But it is not very rare for there to be little but malaise, anorexia and fretfulness; and babies, of course, may be too young to indicate the site of pain. One of the commonest mistakes in medicine, simply because the condition is not considered and the ears are not examined, is to miss otitis media in a young child, and it *does* make one feel a fool! *Deafness* is seldom severe, apart from influenza. *Tenderness* and even a slight *œdema* over the mastoid antrum are not at all uncommon, especially in the young child, in the first days of otitis media. Occasionally one sees vertigo, nystagmus, facial paralysis or meningism quite early, before the membrane perforates (treatment—myringotomy). The membrane may appear quite normal; much more often it is pink, or even scarlet, or showing purple bullæ, or bulging. Remember that the obliquity of the membrane is greater in a young child than adult life, and in the new-born the membrane looks downwards and forward rather than outwards.

Differential Diagnosis.

The condition most likely to cause confusion is a furuncle, particularly if accompanied by post-aural lymphadenitis. The immediate history is often helpful in diagnosis, and inspection of the meatus should reveal any *œdema* there, and possibly the membrane and hearing will be found normal. Pain in the ear may be occasioned by herpes oticus (the vesicle on the membrane looks very like a perforation), dental caries, sphenoidal sinusitis (I have had a case at four years old), or, rarely, pneumonia; it may be the first sign of meningitis (tuberculous or meningococcal).

Complications.

Complications occasionally arise quite early, even before the membrane perforates. More commonly such an event occurs some days after this has happened, heralded by return of pain and fever. Most commonly of all, a complication accompanies an acute exacerbation in chronic otitis. Nephritis is not very rare. Facial paralysis is somewhat less so. (Paralysis of the external rectus has no diagnostic significance.) Extradural abscess shows itself only by recrudescence of symptoms that were subsiding.

Lateral sinus thrombosis in the invasion stage the same ; but in the disintegration stage a rigor occurs—a rigor with otitis media nearly always has this significance. Meningitis requires the most prompt and vigorous handling—every hour wasted before operation lessens the patient's chance ; recovery can take place even though living streptococci are found in the C.S.F. Labyrinthitis is typically heralded by vertigo and vomiting, but children seem little discomposed by vertigo as compared with adults, and if confined to bed may make no complaint, even when brisk spontaneous nystagmus is present ; examine for this if the child lies all the time with the bad ear uppermost. Brain abscess is, fortunately, somewhat rare in children.

Treatment.

The multiplication of aural clinics has tended to foster the belief that the treatment of otitis media is a mystery, requiring special equipment or extraordinary ability. While such clinics properly cater for those who are unable to pay for treatment, there is no reason at all why the family doctor, with the assistance of even moderately intelligent parents or nurse, should not deal perfectly successfully with every case of otitis media, except possibly the very few that need operative treatment. Certain points should be kept in mind : First, of all those cases (doubtless chiefly the milder) that receive no treatment at all, the great majority recover perfectly ; the test of any method is not its many successes, but the rarity of failures and complications.

Every attack of otitis media should be envisaged as involving the whole middle ear tract, including the antrum and any cells of the mastoid, from the very first ; we cannot “ stop the spread of infection to the mastoid,” but we can do much to limit damage and assist resolution. The organisms that are important are not those floating about in the discharge, but those buried well beneath the surface of the inflamed mucous membrane, inaccessible to all medication that will not damage the latter. Even were this not so, and in those cases where the perforation is large, meatal medication can have little direct effect on the bulk of the mucous membrane (*cf.* Figs. 2, 4), especially when we remember that the attic is almost filled with ossicles, ligaments and swollen mucosa (Fig. 5), and the spaces that remain are occupied by thick purulent or mucoid discharge. Our object must be to promote drainage while doing as little harm as possible to the mucous membrane. We

can thus formulate two sound rules in the treatment of all otitis media : *Never* put hydrogen peroxide in an ear ; and *never* syringe a running ear.

Acute Stage.

General Treatment.—Bed, calomel, mist. ammon. acetat. or aspirin (powdered with a little sugar) ; water or glucose lemonade *ad lib.* ; only if the child is hungry : soup, porridge, milk pudding, bread and jam, or honey.

If the membrane has not perforated instil into the ear every four hours, as warm as can be tolerated, drops of glycerine containing 2 per cent. phenol (the B.P. preparation is too strong)—it is both analgesic and disinfectant. Tr. opii is also effective, as papaverine is an obtundent ; but it should not be used for babies, as the inflamed membrane is capable of absorption. The ear may be laid on a hot-water bottle. Avoid poultices. Dover powder is useful if the pain is bad.

Myringotomy.—There is some evidence that the incision made artificially heals more rapidly and with less risk to hearing than a natural perforation of the membrane. The indications for operation are (1) severe toxæmia or deafness ; vertigo, or signs of meningeal irritation, any of which is occasionally seen quite at the outset of the disease ; (2) pain—one sleepless night, or severe pain lasting six hours after the start ; (3) fever, or a bulging membrane more than forty-eight hours after the beginning of the attack. A general anæsthetic is essential.

When the ear is discharging the glycerine and phenol treatment outlined above will be suitable during the first three days. In addition, before instilling the drops, clean the ear with dry wool pencils (*see below*) ; and after each instillation cover the ear with a sterile dressing. Do not “plug” the ear with wool, and, above all, *never syringe a discharging ear*. After three days, pain, cedema and fever should have disappeared ; if not, operative treatment will probably be necessary sooner or later. If all is well, the child need not be confined to bed, but should remain in a warm room (except in the warmest weather). The glycerine drops may be stopped, and instead crystalline magnesium sulphate in fine powder dusted freely into the meatus after each cleaning ; the discharge rapidly diminishes, and treatment twice a day is soon sufficient ; with this a large blob of wool in the bowl of the concha may be substituted for the dressing by day if the child can be trusted not

to interfere with it. Under this régime the discharge usually ceases, and hearing returns to normal in from four to twenty days—about ten is the average. Once a week the ear should be inflated by Politzer's method. The general condition should receive consideration, and any necessary treatment begun; iron is often valuable. If at the end of four weeks the discharge has not ceased, or is at least not obviously just about to cease, the question arises whether infection is kept up (a) from the naso-pharynx, or (b) from the mastoid cells; no further time should be allowed to pass before these questions have been answered. Children possess great powers of recovery, but the longer infection of the middle ear is allowed to persist, the greater becomes the risk of some permanent damage to the hearing. And during the whole time that discharge occurs, even though the amount be trivial, the temperature should be taken morning and evening, and frequent careful examination made for œdema or tenderness in the mastoid region.

Mastoid Operation.—The indications for mastoidotomy are: (1) Pain or fever or tenderness persisting unrelieved more than forty-eight hours from the onset of discharge. (2) Œdema persisting thus long, or considerable œdema at any time; the swelling is most commonly seen behind the ear or in the posterior wall of the meatus, but it may be in front of or above the ear, or in the neck below the mastoid tip, or (rarely) in the lateral wall of the pharynx. (3) Headache or signs of complications (outlined above). (4) Persistent discharge, malaise, anorexia, wasting. Remember that serious infection of the mastoid, or serious complications such as meningitis, can occur *before the membrane has perforated*.

Chronic Stage.

If when the patient is first seen the ear has been discharging more than a month, attention should be directed to the question of the general health and the probability of naso-pharyngeal disease, and appropriate treatment begun. (It is assumed that the ear is painless; acute exacerbations of chronic disease should be treated on the lines of acute otitis media.) The ear should be cleaned out with mops of dry wool, and the membrane inspected to see the size and position of the perforation. There may be granulations growing from the edge, and it sometimes shortens treatment to cauterise such with 20 per cent. silver nitrate on a very fine wool probe. An aural " polypus " nearly always indicates operative treatment.

The parents or nurse must now be taught to make a good aural mop, a thing few know how to do. A thin pencil of wool is needed, not a hard round blob. Take a thin piece of the best wool (cheap fluff is quite useless) about one inch by two, and hold it on the left index finger, palm up. Place the wool-carrier (match or orange stick) on this, in the line with the long axis, and projecting half an inch over the edge. Fold the wool round the stick with the left thumb, and holding it thus, rotate the stick so that the wool is firmly twisted to it. Gradually withdraw the wool and stick, continuing the pressure and rotation to form the wool into a thin pencil, leaving the end fluffy. Plunge this straight down the meatus to the membrane, holding the pinna outward to straighten the passage, and withdraw the wool without twiddling it round. Repeat this until the wool comes away clean. Half fill the cleaned meatus with finely powdered crystalline magnesium sulphate, and protect the ear with a blob of wool in the bowl of the concha. As a rule treatment once a day suffices to keep the ear clean ; it is more important to have the meatus properly cleaned than to have frequent applications of the powder.

As soon as the discharge diminishes to a trace, one may substitute iodo-boric powder for the magnesium sulphate.

R	Iodi	gr. 5
	Ac. boric	ad unciam.

Rub together dry into a fine uniform powder.

This may be dusted into the ear, or, better, insufflated by means of a powder blower. It soon becomes unnecessary to treat the ear more than twice a week, and healing follows. While treatment on these lines is going on it is a good plan to inspect the ear at least once a week (at first) to ensure that the meatus is being cleaned, and that no mass of powder is blocking it. (Should this occur, it means that discharge has ceased ; the powder can be removed by wiping, after instillation of two drops of water, but parents should not be allowed to do this : their duty is to keep the ear absolutely dry.) Advantage should be taken of this weekly visit to inflate the ear.

The child may lead a normal life, with special care to avoid exposure to wet and cold, but must not visit bathing pools ; sun bathing, or sea or river bathing if the water is really warm, may be allowed, provided that the ears are protected from the entry of

water by means of fibrous plasticine. The same substance may be used in cases with healed open perforation, or after a radical mastoid operation with a healed cavity.

At the end of one month's treatment the discharge should be clear, inoffensive and of small quantity, and after six weeks healing should have occurred, or be obviously in sight. If after two months at most this stage is not reached, it is almost certain that there is chronic disease of the mastoid (or ossicles), and probably cholesteatoma. The question of operative treatment should not be further postponed; it is unpardonable to allow month after month to slip by while the child's chances of useful hearing grow less and less simply from a refusal to face the fact that treatment through the meatus is not succeeding. The ear is the organ of hearing, not a drainage tube for a diseased mastoid.

CHAPTER XXIX.

THE TREATMENT OF RHEUMATOID ARTHRITIS IN GENERAL PRACTICE

BY

W. S. C. COPEMAN, M.A., M.B., M.R.C.P.

Physician to the British Red Cross Society's Clinic for Rheumatism, Peto Place, London; Assistant Physician, Children's Department, West London Hospital, and Hospital of St. John and Elizabeth; Consultant, L.C.C.

THE rheumatic group of diseases includes nine or ten definite clinical syndromes, possibly quite unrelated to one another, and ranging from Still's disease to sciatica. It is, however, a matter of convenience, as well as of tradition, to retain this arrangement, since no very good purpose would appear to be served at this stage by the general adoption of those more elaborate classifications which are proposed from time to time. The rheumatic group may be regarded as the Cinderella of medicine by virtue of the increasing attention which it is at last beginning to attract after centuries of neglect.

Rheumatoid arthritis, which is the subject of this paper, is no longer used as a generic term for all those members of the rheumatic group whose ravages fall chiefly upon the joints. The term is now used for a very definite clinical syndrome whose cause is still unknown, although a certain proportion of cases seem to be due to focal infection.

Definition.

Rheumatoid arthritis is a progressive, although often remittent and crippling, form of inflammation affecting generally the small joints first and spreading subsequently to the larger ones. It ends, if unchecked, in fibrous or bony ankylosis. It occurs more frequently in women than in men, and attacks them chiefly during the child-bearing period of life, either in the early twenties, or near the menopause.

The earliest signs are generally a bilateral symmetrical wasting of the small muscles of the hands, certain menstrual irregularities, and a mild secondary anæmia. Intermittent swelling of the centre joints of the two middle fingers is also very common. The course of the disease is generally subacute, it may be with a low grade pyrexia. Unless treatment is started early, contraction deformities in all the affected joints, with great pain, are the rule. The disease runs its course more rapidly in the younger patients, and girls of eighteen to twenty, apparently previously healthy, are frequently seen who have reached this endpoint within a year from the onset of the disease.

Causes of Failure.

It is a matter of common experience that this type of case is but little influenced directly by ordinary drug therapy alone, and this knowledge has led many general practitioners to consider the disease as being either hopeless (and, therefore, to be left to run its course) or outside their sphere of action (which also leads to its neglect). Certain further causes for the bad results often obtained may also be mentioned. The doctor may for some reason pin his faith to a certain form of treatment—it may perhaps be vaccines—and, despite all local or general indications for auxiliary forms of therapy, relentlessly pursue the appointed course of injections, “looking neither to the right nor to the left.” No rule-of-thumb method is likely to succeed in such a complex syndrome, and success will only come by the intelligent combination of appropriate remedies at the correct moment for each.

Another cause of failure is often the exclusive and uncritical employment of the latest proprietary anti-rheumatic medicine. These products are generally only pain-relieving drugs with long formulæ, and disillusionment generally results shortly. The expectation of quick results and the all too common advice to the patient to “walk it off” are two further barriers to success. One of the greatest obstacles seems, however, to be the widespread neglect of the fact that the body can be influenced as powerfully from the outside as from the inside by means of physical medicine.

Preliminary Considerations.

It is important to remember that in rheumatoid arthritis, over-treatment, even when this is on the right lines, is as harmful as under-treatment. These sufferers are generally of frail physique

and of very poor bodily resistance. Intelligent co-operation with the patient is a *sine qua non* in this as in all chronic diseases which tend to be progressive, and a planned programme of therapy on general lines is therefore necessary, and should be explained carefully if the patient be of sufficient intelligence. The patient's life must also be regulated, since no treatment is likely to be successful in the presence of chronic fatigue. It seems reasonable to treat these cases, so far as is practical in the individual case, on the same lines as chronic tuberculous patients, with a maximum of fresh air and light, and an optimum mixed diet.

Treatment.

The search for and removal of septic foci is a *sine qua non*. Whilst admitting the wisdom and necessity for this as a preparatory measure, it has been found wise not to encourage unduly sanguine hopes in the patient's mind as to its actual curative value.

Further treatment will be divisible into two definite categories : (1) local measures ; (2) general measures ; and these should always be considered separately by the medical man treating these cases.

(1) LOCAL MEASURES.—These will include procedures for (a) relieving pain ; (b) giving rest ; (c) restoring function to affected joints, or, if this is impossible, preventing permanent deformity.

(a) *Relieving Pain*.—Drugs useful for this reason are numerous. Aspirin seems still to be the best in a majority of cases. The action of aspirin is a local one at the affected area, and it is often well to reinforce this with some centrally acting drug of the phenazone group, such as pyramidon or novalgin. Sodium salicylate is generally useless, although traditional. Occasionally recourse has to be had to the opium derivatives, when a prescription of aspirin, gr. v, caffeine, gr. x, and codeine phosphate, gr. 1/6, will be found useful, especially at night when sleep is interfered with. The atophan group is often used, but should be carefully controlled.

Heat applied locally to the area of pain generally gives relief, and should be ordered. Methods of doing this without special apparatus are by poultices, antiphlogistin, mud packs, hot-water baths, or hot paraffin wax painted round the joint (moist heat) ; exposure to a gas-fire (which contains infra-red rays), a small radiant-heat lamp, hot-water bottles, or electric pads which can be plugged into the light circuit (dry heat).

(b) *Giving Rest*.—When a joint is movable but painful, it is wise to rest it, to avoid unnecessary pain. This may be done by means of splints, sandbags, or elastic or other bandages. Full movement must, however, be insisted upon at least once every day in order that ankylosis may not be facilitated. Extra rest is also necessary in these cases for the patient as a whole, since a fatigue factor, either mental or physical, is present in most cases and must not be overlooked. Tiring forms of treatment, such as baths or vigorous massage, are contraindicated in the active stage for the same reason.

(c) *Restoring Function to Affected Joints*, or, if that is impossible, preventing permanent deformity. The removal of pain will sometimes restore the movement in an affected joint; in most cases, however, it is necessary to splint the joint in good position for at least a few hours daily until the activity of the disease has subsided. After this the muscles supporting and working the joint must be redeveloped by exercises if possible, or, failing these, by such electrical means as Faradism from the Morton Smart Coil, or interrupted galvanism. Until this period arrives, it is wise to order regular gentle massage to these muscles, to preserve the integrity of their blood supply. It must be remembered, however, that no amount of massage alone will actually build muscle.

If a complete ankylosis of the joint has already occurred in the course of this disease, little can usually be done, since manipulation is not by any means indicated in the majority of cases. Where there is merely defective movement, however, much can be done with encouragement of the patient gradually to increase the range, and, with the help of a trained C.S.M.M.G. masseuse, if this is possible. Some form of local heat applied to the joint for thirty to forty minutes is always a useful preparatory measure, since it loosens the tissues and relieves pain. Diathermy is a good source of such heat where it is available, whilst if there be much effusion, ionisation with almost any of the usual drugs is useful. Operative measures are only to be considered as a last resort for straightening limbs whose joints are contracted and ankylosed.

(2) *GENERAL MEASURES*.—This important section of treatment is frequently neglected in the case of rheumatoid arthritis. It is, however, essential, and must be directed towards increasing the resistance of the body to the progress of the disease. The following

are amongst the methods of doing this : (a) By increasing the patient's weight ; (b) by stimulating the skin without fatiguing the patient ; (c) by increasing the rate of metabolism and stimulating the endocrine glands.

(a) To increase the patient's weight in the course of this disease is by no means easy. These patients are generally about a stone under weight, and dietetic methods unaided are seldom successful. My own practice is to place them on a diet of high caloric value, and to administer a daily injection of 15 units of insulin before the midday meal for periods of about six weeks at a time. There seems to be no need to restrict the diet of these patients in meat, etc., as is traditional, although twenty-four hours on nothing but fruit-juice once a month or so is often beneficial. The majority of these patients react badly to prolonged dieting or starving.

(b) To stimulate the skin is the aim of many forms of physical treatment, and those methods which achieve this end without causing fatigue to the patient are all indicated in rheumatoid arthritis. The skin appears to be very intimately connected with both the heat-regulating mechanism and the biological defence mechanism of the body, and is almost invariably found to be inactive in sufferers from this disease. Chief amongst these methods are ultra-violet light and light massage. If these patients be treated somewhat on sanatorium lines, additional stimulation will result from both air currents and sun.

(c) To increase the metabolism of the body and the activity of the endocrine glands is possibly a counsel of perfection. It is possible that both those objects are achieved in some measure, however, by stimulating the skin, as has been described above. Small doses of vaccines, of iodine, or of thyroid gland, are sometimes of use, and the latter may be combined with ovarian extract. Injections of certain salts of the rarer metals, such as gold, are being extensively used in France, and considerable success is reported. Small doses of potassium permanganate have also been advised for this purpose in view of the intense oxidising power of this drug. Direct stimulation of the ovaries by means of an electrical diathermy current passed between a vaginal electrode and a pad over the abdomen, has given excellent results in selected cases, as has also the writer's method of blood transfusion for checking the activity of the disease in early cases.

Prognosis.

The prognosis in this disease depends almost directly upon the vision and application of the medical man treating each particular patient. Certain cases (about 15 per cent.) will respond to no method ; but the outlook in the average case is about twice as good as it was thirty years ago if sufficient time and trouble are devoted to its direction. Gross deformity should never occur in modern practice.

CHAPTER XXX.

INFECTIONS OF THE HAND

BY

R. M. HANDFIELD-JONES, M.S., F.R.C.S.

Surgeon to St. Mary's Hospital, London.

THE majority of severe infections in the hand have their origin in trivial injuries or minor sepsis in the fingers, and if these conditions are promptly and adequately treated many crippling deformities of the hand would be prevented.

Prophylactic Treatment.

The immediate attention to small cuts, pricks or scratches is of importance. Contrary to all expectation, bleeding should be actively encouraged, as this will wash organisms out of the depth of the wound. If the finger is dirty it should not be washed or scrubbed, as this can only lead to infection being rubbed into the wound. After three minutes' active bleeding the finger is immersed in a bath of iodine for one minute (the usual dab with a swab is quite futile), and a sterile dressing applied. The finger is then inspected at six-hour intervals.

The Infection in the Terminal Segment of the Finger.

The term "distal pulp" will be used to define the fatty cutaneous pad on the palmar surface of the terminal segment. Its anatomy must be understood, otherwise the old mistakes in treatment will be persisted in, and an unnecessary number of severe infections follow. The distal pulp is a closed compartment, as the deep fascia follows the flexor profundus tendon and gains attachment to the terminal phalanx distal to the epiphysis. The compartment is bounded by skin and by the phalanx, and is divided into a number (usually fifteen) of pockets by fibrous septa which run longitudinally from the skin to the periosteum. Each pocket contains fat and sweat glands, the ducts of which reach the surface, providing a path for infection without actual injury. The symptoms and

signs are those of local inflammation. There is usually the history of a slight injury a few hours before, and the patient then becomes aware of a pricking feeling in the finger end and some soreness, and during the night a throbbing pain of rapidly increasing severity awakens him. The distal pulp will be swollen, red and tense, and very tender; in the early stages the signs may be localised to one side around the portal of infection, but soon the whole finger end is red and swollen. The tenseness and tenderness are the really important signs, and only when the destruction of tissue is advanced does fluctuation become evident. Treatment consists of an immediate incision. The common mistake is the use of the median incision, which can only open one of the fifteen longitudinal pockets, and there can be no guarantee that this incision is opening the infected pocket. The lateral incision, by dividing the fibrous septa, ensures that the infected pocket is entered. In early cases one lateral incision on the side primarily infected will suffice, but in advanced cases a second incision must be made, and through-and-through drainage obtained. In very advanced examples the two cuts may be joined up by prolonging them over the end of the finger.

Paronychia is an infection around the edge of the nail arising from an infected "hangnail," leading to a red and swollen skin margin around the nail (the so-called "runaround"). There is a general lack of appreciation of the pathology. The pus is not between the skin and the nail, but between the nail and its bed; consequently a simple skin incision does not drain the infected area. Treatment consists of an incision which continues the line of the lateral margin of the cuticle, and which is continued half way to the distal extension crease. An exactly similar incision on the other side of the finger produces a flap which, on being raised, exposes the nail root. One blade of a pair of pointed scissors is pushed beneath the nail, which is divided so that the nail root can be removed, while the exposed nail lies firmly in position on its matrix. A strip of rubber tissue maintains drainage for thirty-six hours, and the flap heals rapidly. As the new nail grows only a small slit of matrix is left exposed; thus is the long and tiresome convalescence of a complete nail removal avoided.

Acute Lymphangitis.

The palmar surface of the fingers and palm contain a dense capillary network of superficial lymphatics. At the wrist all these

superficial tributaries are gathered into a number (between twenty-four and thirty) well defined vessels, which travel upwards to the axilla. Acute lymphangitis is an infection of these lymphatics, and is usually of streptococcal origin. The site of infection is usually in the terminal segment of the finger, and organisms gain entrance through a trivial prick or scratch. The infection is confined to the lymphatics, and spreads rapidly up into the palm and forearm, where the infected vessels are obvious as bright red lines starting at the wrist, and going upwards towards the elbow.

The symptoms are somewhat similar in the early stages to the distal pulp infection. A few hours after injury the patient complains of a feeling of chill, and there may be a rigor. There is pain and swelling in the finger end, but the pain is not throbbing in character, and the swelling is not so marked as in pulp infections. The constitutional symptoms are unexpectedly grave for so apparently trivial a local lesion, and, in addition, the tell-tale "red lines" in the forearm announce the diagnosis.

There are four degrees of acute lymphangitis, namely: (1) Without local complications; (2) with local complications, *e.g.*, an abscess at the site of infection; (3) with severe systemic involvement and severe local complications, *e.g.*, tendon sheath infections; (4) the fulminating type, which is always fatal in spite of any treatment.

There is nothing in medical practice so terrible as the death of a patient who pricked his finger on a rose thorn or splinter of wood only forty-eight hours before. It is imperative that every practitioner should realise that an incision in a case of acute lymphangitis will probably convert a mild case into a severe or fatal one. In the presence of "red lines" in the forearm, no incision should ever be made until a second opinion has been obtained.

The treatment is local and general. Local treatment consists in swathing the whole arm, from finger-tips to shoulder, in a linseed poultice, which is to be changed every four hours. It is surrounded by a large covering of wool, and lightly bandaged. The patient is to be kept in bed and carefully nursed. The slight cases will clear up as dramatically as they began; but if the red lines are advancing, the swelling increasing and spreading to the forearm, the practitioner in charge of the case will know that a severe infection is present, and that the danger of a general septicæmia is imminent. In these cases a second opinion should be obtained at once. There is nothing which calls for more skill, more unremitting care and

more devoted nursing than septicæmia. The details of general treatment cannot be dealt with here, but it is absolutely imperative that no incision should ever be made in acute lymphangitis until an obvious collection of pus is present.

Collar-Stud Abscess in the Palm.

Infection sometimes gets into the subcutaneous tissues beneath the hard callosities which form in the palm at the base of the fingers. A brawny red swelling appears at the distal part of the palm at the base of a finger, and spreads to the back of the hand *via* the web on one or either side of the finger. An abscess forms which has one pocket beneath the skin and another beneath the palmar fascia, the two being connected by a small channel. If the deeper pocket is not opened, pus may readily spread into the middle palmar space, and a severe infection follow.

Treatment consists in an incision through the brawny swelling in the long axis of the hand. The pus is swabbed away, and a pair of sinus forceps enters and dilates the narrow passage into the deeper pocket. If necessary, drainage can be made more efficient by a small cut in the web on the dorsum of the hand.

Tendon Sheath Infections.

There is some variation in the normal anatomy of the tendon sheaths in the fingers and hand, but the following describes the most common arrangement. The sheaths of the index, middle and ring fingers extend from a point just distal to the distal interphalangeal joint into the palm, about a thumb's breadth proximal to the web. The sheath in the little finger does not end blindly in the palm, but opens out into a synovial sac known as the ulnar bursa. This reaches the base of the middle metacarpal, and passes upwards under the anterior annular ligament, and here receives the tendons of the middle, ring and index fingers. The sheath of the flexor longus pollicis extends from the base of the distal phalanx through the thenar muscles under the anterior annular ligament. It is here known as the radial bursa. The two bursæ end on the anterior surface of the pronator quadratus muscles.

Infection may reach the sheaths either as the result of gross crushing injuries, by which the sheath is opened, or as a complication of other conditions, *e.g.*, distal pulp infections or lymphangitis, in which the organisms are carried to the sheaths by the lymphatics. The importance of sheath infections cannot be over-estimated.

Delayed diagnosis and improper treatment may lead to crippling of the whole hand, and imperfect treatment will result in one stiff finger. It must be understood that skilled treatment results in restoration to perfect function.

The symptoms and signs are: (1) Symmetrical enlargement of the entire finger, the swelling extending on the dorsum of the hand and into the bases of the contiguous fingers. (2) Exquisite tenderness over the course of the sheath. (3) Excruciating pain on complete extension of the finger, most marked at the terminal end of the sheath in the palm. Gentle movement of the finger is tolerated with considerable anxiety until full extension is approached. (4) The finger at rest will be held in moderate flexion. This description is true of all the five digits, but in the case of the thumb and little finger the area of tenderness will spread into the palm corresponding to the extent of the bursæ, and there will be great swelling of the hand. The patient's general condition will be poor, the temperature high, and possibly a rigor may occur.

Treatment is based on early diagnosis. The danger lies in the loss of the vitality of the tendon and its necrosis, leaving a crippled finger. Even after early incision another danger consists of the introduction of a secondary infection into the wound. Most acute tenosynovitis lesions are streptococcal, and early and careful treatment will result in a perfectly moving finger. If, however, staphylococci are allowed to gain entrance, an adherent tendon is certain to follow. Delayed diagnosis and imperfect treatment may lead to severe crippling of the whole hand. The importance of this condition need be stressed no further than to ask each medical practitioner to picture his own feelings if he had an acute tendon sheath infection. The surgery of such conditions can never belong to the "minor" class. In brief, treatment consists of drainage of the sheath by an incision, which has so often in the past been made in the mid-line of the finger. It is almost impossible to save a tendon from sloughing with this incision. The cut should be made laterally, and the vaginal ligaments opposite the interphalangeal joints should not be divided.

The Fascial Spaces of the Palm.

The fascial spaces in the palm, which are of surgical importance, are the middle palmar or the thenar spaces. The middle palmar space lies behind the flexor tendons and lumbricals of the little,

ring and middle fingers, and in front of the metacarpal bones and interosseous muscles. It reaches the central line of the shaft of the middle metacarpal, along which a fibrous septum separates it from the thenar space. The thenar space lies behind the flexor tendons and lumbrical of the index finger, and in front of the adductor transversus pollicis muscle. It reaches the central line of the middle metacarpal and extends into the thenar muscles. The spaces are usually infected by spread of pus from other lesions in the fingers or hand. The signs are great swelling and exquisite tenderness over the surface area of the spaces concerned. Such an infection denotes a very serious condition of the hand, for which surgical advice is urgently needed. The treatment consists of an incision into the space concerned. The middle palmar space is reached from the palm by an incision which will open up the lumbrical canal of the ring or little finger. The thenar space is opened by an incision on the back of the hand along the shaft of the index metacarpal, which exposes the lower border of the adductor transversus pollicis in front of which the space lies.

Operative Technique and After-Treatment.

As I have reached the end of the space allotted to me, I must present this section in skeleton form in the way of advice. (1) Always use a tourniquet, preferably the arm compressor of a sphygmomanometer. (2) Always use a general anæsthetic; never under any circumstances an ethyl chloride spray locally. If a local anæsthetic is used, it must be a nerve block. (3) Never make an incision until the presence and the exact anatomical location of pus has been decided upon. (4) Never make an incision on the dorsum of the hand until every possible site of pus in the palm has been eliminated. (5) Great harm can be done by too small incisions, very little by too long. (6) Never use drainage tubes, only wicks of rubber tissue. (7) Hot fomentations need never be used after forty-eight hours, then small paraffin and flavine dressings. (8) Concentrate on the restoration of movement from the very beginning. Make patients keep the fingers moving, and this is most easily done in hot baths, either iodine, or, better still, hot dry air baths. With the same purpose keep the dressings as small as possible, and loosely bandaged. (9) Finally, do not regard septic fingers and hands as trivial things; they may be, but on the other hand, they may be the beginning of a useless crippled hand.

CHAPTER XXXI.

THE TREATMENT OF DIGESTIVE DISTURBANCES IN THE BREAST-FED INFANT

BY

ROBERT E. STEEN, M.D., F.R.C.P.I.

Physician to the Meath Hospital, and to the National Children's
Hospital, Dublin.

EVERY baby should have the advantage of breast-feeding for at least the first few months of its life, unless there is some special contraindication. The advantages of breast milk to the child are : (1) no artificial food can compare with it in the matter of digestibility ; (2) it is almost sterile, and this is of particular importance during the late summer months, when summer diarrhoea is at its height ; (3) it possesses immune substances which it transfers from the mother to the child. Statistics show that the mortality in breast-fed children is one-fifth of that in other children in the first year of life. Other points might be added, but these will be sufficient to show that it is of the greatest importance to the young baby that the breast milk should be conserved, and that premature weaning should be avoided unless there is some very definite indication which we are unable to remove. It must be admitted, however, that there will always remain a large number of mothers who for one good reason or another cannot nurse their babies.

In the present article it will be only possible to consider in any detail the treatment of those disturbances in the breast-fed infant which are due to faults in the feeding. The practitioner, however, who is called in to see a breast-fed infant who is suffering from a digestive disturbance should first make sure that the real cause of the trouble is not some parenteral condition, *e.g.*, acute otitis media, pyelitis, etc., since even the " common cold " is a frequent

cause of a digestive upset in a baby. The treatment of such an upset is chiefly a matter of treating the cause.

When those conditions are considered where the upset of the digestion is primary, the possibility of infection, *i.e.*, an acute infective gastro-enteritis, should be borne in mind. Mild infections are not very uncommon, but fortunately severe infections of the bowel are extremely rare in the breast-fed baby compared with the artificially fed. When an acute infective gastro-enteritis occurs it is usually, as in the case of artificially-fed babies, during the summer months, especially the late summer months, and is known as "summer diarrhoea." Its treatment is similar to that in the artificially-fed case. Skimmed breast milk is usually the feed of choice, just as a skimmed dried milk is the feed of choice in an artificially-fed infant. The importance of supplying fluid when dehydration is present cannot be over-estimated. This may be given by mouth in the form of half strength normal saline (a level teaspoonful of salt in a pint of boiled water). The salt appears to be of great value in assisting the tissues to retain the fluid. It may be given freely as a drink from time to time, or several ounces may be left in the stomach after it has been washed out. In a severe case it should also be given in the form of 2 per cent. glucose saline subcutaneously, intraperitoneally, or intravenously *via* the fontanelle. The addition of a little insulin (five or six units to the pint) fixes the glucose in the tissues and prevents loss in the urine. One can usually get in 8-10 oz. by the subcutaneous route, and on the whole I prefer this route for routine purposes; less than half that amount may cause respiratory embarrassment by the intraperitoneal route, but it has the great merit of being a rapid procedure, and though it is largely employed, and I have used it myself on many occasions, I have never heard of an instance where the gut was punctured. I do not think that the intravenous route will be often called for, but I believe that I have saved lives by this means when the circulation was so enfeebled that fluid by other routes would not have been absorbed.

The practitioner should also keep in the back of his mind the possibility of the dyspepsia being due to gross organic disease, *e.g.*, congenital pyloric stenosis, or those functional gastric disorders such as habitual vomiting, pylorospasm, rumination, or the like. The treatment of the latter is outside the scope of this paper, but

the value of Rammstedt's operation for congenital pyloric stenosis may be mentioned.

When everything has been considered, however, there is no doubt that the most frequent cause of digestive disturbances in the breast-fed infant is faulty feeding. *It should, nevertheless, be clearly realised by doctors that the composition of the breast milk is rarely at fault.* It is true that the composition of the mother's milk varies in different individuals, in the same individual at different feeds, and at different stages of the same feed, and in the same individual at different periods of lactation; but in spite of such variations, breast milk is so digestible that it rarely disagrees *per se*. For this reason most physicians have abandoned the practice, except in very rare instances, of having samples of milk sent for biochemical examination. It should be clearly realised that in the large majority of cases faults in breast-feeding are due to (1) irregular feeding; (2) underfeeding; (3) overfeeding. To wean a baby because the milk is "blue," or because it is "thin" (especially when the sample of milk inspected is the first withdrawn), or because of "windy milk" (which is nearly always due to air-swallowing from sucking at an empty breast), is unscientific therapy.

Irregular Feeding.—Irregular and too frequent feeds are a common cause of trouble. "Feeding the baby by the clock" will correct this. For example, if it is decided to feed three-hourly, the child should be fed at 6, 9, 12, 3, 6, and 10 o'clock; or if the feed is to be four-hourly, it should be given at 6, 10, 2, 6 and 10 o'clock. Night feeds, except in very weak and undernourished infants, should be as far as possible avoided. If the child is awake it may be given a little boiled water.

Underfeeding.—This is a most prolific cause of difficulties with the breast-fed infant. The correct diagnosis is often missed, and the milk itself incriminated because it is not realised that an underfed baby may have as a symptom vomiting (usually from air swallowing, the result of sucking at an empty breast), or diarrhoea ("inanition diarrhoea"), though more usually at the beginning it has the constipation of starvation, *i.e.*, a tiny green stool consisting chiefly of mucus, and passed at very infrequent intervals, *e.g.*, 36 to 48 hours. Even in the latter instance the mother may give a history of diarrhoea, basing her judgment on the colour rather than the frequency of the stools. The treatment of underfeeding may be

grouped into, firstly, the treatment of faults on the part of the child, and, in the second place, the treatment of faults on the part of the mother.

Treatment of Faults on the Part of the Child.

(1) *Nervousness*.—Some children seem to have hyper-excitable temperaments, and they attack the breast vigorously and almost angrily, and fail altogether to settle down to steady quiet feeding. As a result more air than milk may be swallowed, and digestive disturbances result. The mother is often of the nervous type described below, and she appears to impart some of her nervousness to the child. The treatment consists firstly in firm handling. A capable nurse is a great assistance. Feeding should be carried out in a quiet room, which may with advantage be darkened. The baby may be wrapped firmly in a light shawl to prevent it struggling while feeding. Lastly, the administration of a sedative is of the greatest value. Chloral, gr. $\frac{1}{4}$ to 1, may be given in water twenty minutes before each feed, so that the sedative effect is obtained while the infant is actually at the breast. Cases have been reported where by this procedure the breast milk was doubled in forty-eight hours. Chloral is, of course, particularly well tolerated by the young, and relatively much larger doses may be given to them than one would give to an adult.

A particular form of nervousness is known as the "breast-shy infant." This condition is where the infant shows a dislike to natural feeding, and turns its head away when it is approached to the breast. It is treated on similar lines. Success is often achieved by placing the nipple in the baby's mouth, and squeezing a stream of milk into it. Moistening the nipple with glycerine may also be tried. Feeding at somewhat long intervals is also useful, as the child comes more hungry to the breast.

(2) *Weakness in Sucking*.—The treatment of this is on the same lines as that of a premature baby.

(3) *Respiratory Obstruction*.—Interference with nasal breathing is the commonest form of this, the child being often said to have "snuffles." The infant finds that it has to release the nipple in order to take a breath, and as a result steady quiet feeding is interfered with, and air-swallowing is common, with its attendant disturbances. The treatment consists in running a little alkaline lotion, e.g., a pinch of sod. bicarb. in a little warm water, into both

nostrils, and drying with a tiny pledget of cotton wool. If this fails, a drop of adrenalin, 1 in 1,000, may be instilled into each nostril before nursing. In a very stubborn case the possibility of congenital adenoids must be considered. If present they must be removed.

(4) *Local Conditions*.—Hare lip, cleft palate, stomatitis, cracked lips, etc., need no special comment.

Treatment of Faults on the Part of the Mother.

(1) *Nervousness*.—This is one of the greatest difficulties with which one has to contend. The nervousness not only diminishes the supply of milk, makes her sceptical of ultimate success, but also reacts on the child. The treatment is largely suggestion. She may be told that a certain amount of crying is good for a baby, and makes it expand its lungs. An optimistic attitude should be taken, and any improvement in the weekly gain in weight made much of. A friendly and sympathetic understanding of difficulties is more effective than any amount of argument.

(2) *Local Conditions*.—Retracted nipples, cracked nipples, breast abscess, etc., are dealt with fully in the ordinary text-books, and there is nothing further to add.

(3) *Deficient Secretion of Milk*.—This is probably the most common cause of underfeeding. It is worth emphasising that very little weight can be placed on inspection of the breasts, since often the small flat breast yields abundantly, while leaking breasts may be due to poor muscular tone, and they may contain little milk. The only certain way of making a diagnosis, with the exception of the therapeutic test of weaning the baby, is by performing "test-feeds," *i.e.*, weighing the baby before and after several feeds, and thus estimating the average quantity of milk received per feed.

Measures for Increasing the Quantity of Breast Milk.

(1) *Give Both Breasts at Each Feed*.—Though this procedure is not universally accepted, there is much to commend it. The baby should suckle the breasts for a much shorter time than is usual, otherwise, as a result of sucking at the empty breasts, it fills itself with wind, and its digestion is upset. Three to six minutes is usually ample. In case this period should be thought too little, it should be borne in mind that it has been proved that on an average even the normal breast is half emptied in two minutes, three-quarters of the milk is removed in four, and after eight minutes

very few babies get any milk at all. Mothers often make the mistake of thinking that resting the breast is what is needed in order that they may have longer to fill, and that nothing will be gained by giving both breasts at each feed, since each will now have only half the time to fill. It should therefore be explained to them that *the best stimulus to the secretion of milk is the sucking of the infant*, and that each breast will now be stimulated twice as often in the twenty-four hours.

(2) *Any milk left behind should be expressed and given from spoon or bottle.*—Quite a surprising amount of milk is left behind, even when a hungry baby has finished and appears to have got all that is there. Expression has two advantages: (a) the complete emptying of the breast is, next to the sucking of the infant, the best means of making a breast increase its supply of milk; (b) the extra breast milk will be a valuable addition to its feed.

(3) *Local Applications to the Breast.*—These have some value, their action being on the local circulation, and they also have a valuable psychological effect. (a) *Massage.*—This consists of rubbing and kneading the breasts between the hands, and of squeezing the breast towards the nipple. (b) *Hot and cold sponging.*—This is a well-known valuable stimulant to the circulation. Two basins are necessary, one with cold water, and the other with water as hot as can be comfortably borne. Sponge the breast first with the cold water, and immediately afterwards with the hot. This should be repeated several times, and the whole completed by rubbing with a rough towel. These measures should be carried out two or three times a day after feeds.

(4) *General Health Measures.*—(a) *Diet.*—Provided the diet is well balanced and sufficient for her needs, little or no effect on the mother's milk will result from increasing it. Furthermore, any tendency to overfeeding may upset her digestion. On the other hand, if her diet has been neglected, it will well repay consideration. In a mother who came to me because her twins were not thriving, attention to this point alone caused the weekly increase in their weights to be more than doubled in both cases. (b) *Fluid.*—Plenty of water should be drunk, and one pint of milk daily. Stout also seems sometimes to have a beneficial effect. (c) *Sleep.*—Get to bed as early as possible, and lie down for one or two hours' rest during the early afternoon. (d) *Exercise.*—Some mild out-door

exercise is good, but it should never lead to a feeling of tiredness.
 (e) *Mental rest*.—Exclude all domestic worries.

If after the above measures have been tried for a few days a sufficient improvement is not obtained, and this is likely to be the case except where the deficiency is slight, it will be advisable to have recourse to *complementary feeding*. This consists in the giving of a small artificial feed after the breast feed and any expressed milk have been given. *Supplementary feeding*, i.e., the giving of a complete artificial feed in place of a breast-feed, should never be resorted to, since in most cases where this is done the breast milk rapidly comes to an end. The reason for this is simple. It has already been emphasised that the most important factor in maintaining the secretion of milk is the sucking of the infant. When the milk is already deficient, if a breast-feed is completely replaced by an artificial one, the breasts will receive less stimulation than they were previously receiving, which was already too little, and as each further feed is replaced, the stimulation becomes correspondingly less, until finally there is little or no secretion at all. Indeed, the mother now may feel that her breast-feeds are upsetting the child, and doing it harm, and this is actually the case, because sucking at an empty breast is a useless and harmful pastime for the child. This is, of course, quite a different matter from what is a common practice with some medical men, of replacing the last feed at night by an artificial one. This is said to have certain advantages, and there is no great objection to it where the milk is abundant, because the lack of the extra stimulation will not in such cases be greatly missed.

The complementary feed should be digestible and not too sweet, otherwise the baby loses its appetite for breast milk. The best mixture is one consisting of equal parts of milk and water, with the addition of half a level teaspoonful of sugar to each two ounces of the mixture. The ideal method is to give it after every feed of the day, except the first, which is always a big one from filling of the breasts over night. When this is not practicable, it should be given after the 12, 3 and 6 o'clock feeds, or, if the infant is being fed four-hourly, after the 10, 2 and 6 o'clock feeds. The size of the complementary feed should be judged by the results of the test feeds, and the milk subsequently expressed. Where this is not possible, it must be judged approximately, starting with small amounts and increasing if the child is not satisfied,

or the weight not satisfactory. One or two ounces for each complementary feed is usually sufficient, and it is better to err on the side of giving too little rather than too much, as otherwise the breast milk will tend to fail progressively.

Overfeeding.—This is not such a common cause of difficulties in breast-feeding as under-feeding, since nature provides for the excess of food by slight regurgitation after the feed ("possetting"). When serious disturbances arise it is usually from too frequent feeds rather than from an excessive quantity taken at suitable intervals, and such cases usually respond to lengthening of the intervals between the feeds.

Finally, it may be said that it is only in rare cases that the composition of the milk is at fault. Rare cases, however, have been described where a baby, after failing with three wet nurses, was successfully reared on a peptonised feed. While this is the first thing in the mind of the mother, it should be the last in that of the medical adviser.

CHAPTER XXXII.

THE TREATMENT OF DIARRHŒA IN INFANTS

BY

ROWLAND HILL, M.D., M.R.C.P.

Physician to the Hospital for Sick Children, Belfast.

THE acute diarrhœas of infancy can be classified in three main groups, and it is important in the treatment of an individual case to decide which type of diarrhœa one is dealing with. I propose, therefore, to describe the symptoms which accompany the different types and enable one to form an opinion as to the type present, and thus decide on the correct line of treatment.

(1) *Dyspeptic or Fermentative Diarrhœa*.—This is due probably to some defect either in the quantity or quality of the feeding, especially of the milk. As a rule it is not of infective origin. It is rarely met with in breast-fed children. Simple overfeeding, or excess of any of the normal food constituents—casein, fat or sugar—or decomposition of the food from the use of a dirty feeding bottle, are common causes, though the onset is sometimes precipitated by a chill. The chief symptom is an increase in the number of the stools. Their character varies, depending on the cause. When there is excess of casein in the food the motions are curdy, *i.e.*, they contain visible masses of milk clot. When there is excess of fat in the food the stools tend to be liquid and greasy-looking, and contain here and there throughout them granular masses. On casual observation these masses may be mistaken for small milk clots, but are actually made up of fatty acids, and will be found to dissolve on adding them to equal parts of alcohol and ether. Excess of sugar in the food is not so common a cause of diarrhœa, but in this case the stools are pale and highly acid, and their passage is accompanied by the expulsion of much gas. Whatever the cause, in dyspeptic or fermentative diarrhœa the stools often assume a

green colour from the presence of "biliverdin." Scalding of the parts around the anus is commonly present owing to the acidity of the stools. Frequently the odour of ammonia can be detected from the urine. The abdomen is generally distended, and attacks of "colicky pain" are frequent. The general condition is one of prostration of varying degree, the sinking in of the eyeballs and the anterior fontanelle being evidence of this. Not infrequently the temperature is raised to 99° or 100° F.

In the treatment of this type measures for keeping the child warm should at once be adopted. In the case of a breast-fed child the breast should be withheld for twenty-four hours at least, and in that of an artificially-fed child cow's milk should be stopped completely. Only plain boiled water, albumen water or very weak tea sweetened with saccharin should be given frequently in small quantities. Two or three drops of brandy to each two ounces of whatever fluid is being given will be found useful if there is much restlessness or pain. When first the case is seen it is good practice to give a teaspoonful of castor oil to clear away any irritating debris of food still remaining in the bowel. Thereafter the following mixture should be given every four hours until the stools begin to resume their normal yellow colour: Castor oil, 5 minims; mucilage of acacia, 15 minims; peppermint water, to 1 drachm. As soon as the yellow colour begins to reappear in the stools the previous mixture should be stopped, and the following one should be administered four-hourly: Carbonate of bismuth, 5 grains; calcium carbonate, 7½ grains; tincture of catechu, 5 minims; spirit of chloroform, 2 minims; water, to 1 drachm.

Tincture of opium in 1/2 to 1 minim doses may be given with either of the above mixtures, but should be added to each separate dose of the mixtures, and not dispensed in the whole. When the stools begin to resume their yellow colour, skimmed milk may be substituted for the boiled water, albumen water or weak tea. Sugar, preferably one of the dextrin-maltose preparations, is added in gradually increasing doses until finally a return to an ordinary cow's milk mixture is made.

(2) *Acute Ileo-colitis*.—This condition resembles bacillary dysentery in the adult, and may in some cases be caused by the *Bacillus dysenteriae*. The onset may be sudden, or be preceded by mild diarrhoea for twenty-four or forty-eight hours. The stools at first have the normal yellow colour, then become tinged with

green, and, gradually increasing in frequency, become finally a thin suspension of mucus and blood. With the passage of each stool there are obvious signs of pain and straining, and occasionally prolapse of the rectum. If the case is a mild one the constitutional symptoms are not marked, the temperature rising perhaps to 101° F., but the child remains bright, notwithstanding the frequent stools, and in from ten to fourteen days the diarrhœa begins to lessen, the temperature falls and recovery takes place. In a severe case, however, there is obvious toxæmia with a high temperature, great prostration and rapid dehydration, with stools consisting almost entirely of blood. Ketosis often develops, and death from exhaustion generally follows.

In the treatment of this condition the child should be isolated in a cool, airy room, measures for preserving the body-heat being taken at the same time. All soiled napkins should be removed as soon as possible and placed in some disinfectant solution. The child should be starved for twenty-four hours, but during this period should be encouraged to drink freely of cooled boiled water, barley water or weak tea. At the end of twenty-four hours food may be resumed, but should take the form of a completely skimmed milk, *e.g.*, Cow & Gate separated milk, which is thoroughly skimmed and sterile. This preparation should be made up initially only one-fourth the strength recommended on the maker's tin for the age of the patient, and to each fluid ounce of the reconstituted milk should be added 20 grains of some preparation of casein, *e.g.*, casein, calcium caseinate or protosol. Sugar, which is less well tolerated than protein, may be added in small quantities to the feeds at the end of thirty-six or forty-eight hours, and some preparation of maltose will be found better than cane sugar, lactose or glucose. As the number of stools lessens and the temperature returns to normal, the strength of the milk and the amount of sugar added to each feed can be gradually increased, but it will be found advantageous to continue the addition of the casein preparation to the feeds for some days after the diarrhœa has ceased.

(3) *Acute Gastro-enteritis or Cholera Infantum.*—This form of diarrhœa arises generally in the British Isles after prolonged spells of heat, and may assume epidemic proportions in July, August or September. Children up to the age of five years may be affected, but it is met with most commonly in artificially-fed infants between

the ages of six and eighteen months. Its occurrence is not unconnected with the increase of dust and flies which so often accompanies hot dry weather conditions during the months mentioned, and the difficulty of keeping cow's milk fresh and wholesome in the average working-class house is also a factor. So far no definite micro-organism has been proven to be the *causa causans* of this affection. The onset is often preceded for a day or two by some mild gastro-intestinal disturbance, *e.g.*, abdominal distension or "colicky" pains, but it does occur occasionally in a previously healthy child with great abruptness. The first symptom to appear as a rule is vomiting, followed by a rising temperature, and then the diarrhoea. The stools at first are yellow or brown, then become greenish, and ultimately colourless, containing only shreds made up of epithelial cells, leucocytes and bacterial masses. The vomiting and purging rapidly increase in frequency, the temperature rises to 104° or 105° F., and signs of marked prostration and dehydration quickly show themselves. The face has a markedly pinched appearance, there is sinking-in of the eyeballs, depression of the anterior fontanelle, and lack of elasticity of the skin. In cases which end fatally, purpura and oedema of the feet and legs is often seen.

The treatment of this condition follows the same general lines as that for acute ileo-colitis. The child should be isolated as far as possible in a cool, airy room, and the soiled napkins placed in a disinfectant solution as soon as removed. No food whatever should be given for twenty-four hours, but fluids in the form of plain boiled water, weak tea or N/2 saline as freely as the vomiting permits. The stomach should be washed out with warm water, normal saline or a weak solution of bicarbonate of soda, using either a No. 10 soft rubber catheter or a No. 18 Jaques' oesophageal tube as recommended recently by Young (*The Lancet*, April 1st, 1933). Before the catheter is withdrawn, half an ounce of kaolin suspended in two ounces of warm water is introduced into the stomach, and thereafter at four-hourly intervals the kaolin in doses of two to four drachms in suspension is given either by teaspoon or by re-introducing the tube. Should the vomiting continue so severe as to cause rejection of all fluid given by mouth, or should the purging lead to dehydration, then some other means of introducing fluid into the system should be adopted. Fluids can be given subcutaneously, intravenously or intraperitoneally. The

subcutaneous route does not in my experience permit one to introduce sufficient fluid to overcome the marked dehydration one sees in a desperate case of "cholera infantum." The intravenous route, *via* the superior longitudinal sinus, is satisfactory, but I prefer the intraperitoneal route as being quicker and easier. One has a choice of fluids for injection: normal saline, 5 per cent., glucose in normal saline, or Ringer's solution, quickly prepared from tablets supplied by the manufacturing chemists. The technique of intraperitoneal injection is as follows: First, having made certain that the bladder has recently been emptied, and with due antiseptic preparation of the skin, choose a point on the abdominal wall at the level of the umbilicus and at the outer border of the left rectus, and pinch up a fold of skin and underlying muscle with the finger and thumb of the left hand. Through the fold thrust the needle, unattached, of a 20 c.c. Record syringe downwards towards Poupart's ligament and slightly backwards; attach the syringe, and inject 80 to 100 c.c. of the selected fluid, the fluid being at a t° of 100° F. The operation may be repeated twice or thrice in twenty-four hours. In cases of extreme collapse a stimulant may be called for, and brandy in 5 or 10 minim doses may be given by the mouth, or strychnine, $1/200$ of a grain hypodermically, to a child of six months.

After the preliminary twenty-four hours' starvation a completely skimmed milk, to which should be added some preparation of casein in the proportion of 20 grains to the fluid ounce of milk, should be given. Another form of milk which is valuable is buttermilk, provided it is reasonably fresh. This should be boiled before administration, and then sweetened with some preparation of maltose; a dried preparation of buttermilk is now available in powder form, and makes a valuable substitute for the fresh. Subsequently, the dietetic treatment should follow the same lines as those mentioned in the treatment of ileo-colitis.

CHAPTER XXXIII.

THE MODERN TREATMENT OF NASAL CATARRH

BY

A. L. YATES, M.C., M.D., F.R.C.S.

Assistant Surgeon, Ear and Throat Department, Prince of Wales' Hospital.

THERE are many forms of nasal catarrh which clear up very rapidly with simple forms of treatment. In some of these the best results can be obtained by surgery, in others palliative measures are the best. The history of a case of nasal catarrh is generally of great importance. We can first divide all cases into acute, subacute and chronic.

The acute cases fall into two main groups from the point of view of history. In one the inflammation has produced obstruction to the ostium of the sinus, and an empyema of the sinus has formed. These cases are characterised by pain. It is important to note the presence of previous attacks which show that a case of apparent acute sinusitis is in reality an acute condition grafted on a chronic process. In the second group of nasal inflammation the ostium of the sinus is open. No pain has therefore been present, and the history given is one of fever and malaise which have not yielded to the ordinary forms of treatment. Nasal symptoms are often in this class of case slight.

In subacute and chronic inflammation we get three forms of history. In the one the patient suffers from intermittent pain; in the second, it is the character of the discharge of which the patient complains; sometimes this is excessive in amount, and sometimes it is light, but causes a bad taste in the mouth; and in the third, the history is one of general ill-health, which may be manifested by rheumatism, skin disease or increased bodily or mental fatigue. This last is of considerable importance.

Examination.

Acute Cases.—The headache in the early stages is not markedly severe, but as the case progresses it may become agonising and totally incapacitate the patient from his work. If it is the maxillary antrum which is the site of inflammation this will be dull on transillumination, and the mucous membrane in the middle meatus will be swollen. All practitioners have not, however, the facilities to carry out this examination, but an examination of the fauces gives material information.

In an acute case of sinusitis there can frequently be seen behind the posterior pillar of the fauces a thin white line. This is the main lymphatic from the nose, and, being superficial, is visible through the mucous membrane. The presence of this line gives us the information that examination should be conducted by someone versed in nasal treatment.

In the class of case in which there is no pain, this vessel can still frequently be seen, but sometimes the mucous membrane parallel with the posterior pillar of the fauces is so thickened and congested that it hides the vessel. The presence of this thickening of the mucous membrane is, however, confirmation of the presence of nasal inflammation.

In chronic cases, nasal examination shows swelling of the strip of mucous membrane over which the pus is passing. This swelling is seen in the middle meatus if the frontal anterior ethmoidal or maxillary sinuses are the site of inflammation. In cases of sphenoidal or posterior ethmoidal sinusitis the inflammation is best marked on the upper surface of the middle turbinate.

The diagnosis of the actual sinus is, however, more readily made by a post-nasal examination with a mirror. This also shows a thickening of the mucous membrane in the region over which the pus is passing. Thus, if it is the maxillary antrum, the frontal sinus or the anterior ethmoid cells which are the site of inflammation, the mucous membrane is thickened in front of the Eustachian cushion. If it is the posterior ethmoidal cells which are inflamed, the thickening is seen above the Eustachian cushion; or, if the sphenoidal sinus is the site of inflammation, the pus is passed over the highest and most central portion of the naso-pharynx, and here the mucous membrane is inflamed and thickened.

In the atrophic forms of inflammation these areas of mucous membrane are thickened, but are pallid, and in the hypertrophic

states of inflammation they are thick and red. Transillumination may be a guide to show which antrum is inflamed, or if a frontal sinus is the seat of trouble, but will give no information as to the state of any other sinuses. In chronic states it is good practice to obtain a specimen of the nasal pus in doubtful cases, and examine it beneath the microscope. It is important in this examination to determine the character of the cells and the presence of free micro-organisms.

Treatment.

Acute Cases.—Pain is the symptom which attracts attention when there is obstruction to the opening of an acutely inflamed sinus. In the initial stages the nose should be cocaineised to contract the mucous membrane, and it may be expected that the pain will cease, because the congestion at the opening of the sinus has been removed by the cocaine. The pain will frequently recur, and cocaineisation should be carried out again. If by this treatment the pain is gradually lessened, it should be continued up to, but not longer than, a period of three days. If there is no relief from pain, or if the pain on its recurrence is as great as formerly, or if the pain has not ceased after three days' treatment, the sinus should be washed out when this is possible. If it is not possible to catheterise the sinus, the use of cocaine should be continued as long as it causes an improvement. If, however, the symptoms are increasing in severity it will be necessary to consider the need for operative measures.

In the case of the antrum, the bony wall between it and the nasal cavity will be punctured with a trocar, and liquid paraffin used for lavage. Watery solutions should not be used in acute cases, as they sometimes increase the inflammation. The sphenoid and posterior ethmoid sinuses can be washed out through their normal ostia under cocaine anaesthesia. The frontal sinus can often be washed out by introduction of a soft catheter through its normal ostium. In a few cases it is necessary to open the frontal sinus because the catheter cannot be introduced, but this should be deferred in acute cases whenever possible, as cocaine will generally clear up inflammation of the frontal sinus.

As to the use of cocaine, it is necessary to know how much is delivered by the spray that is employed. Certain makers of sprays—for example, Rogers—show the amount upon the spray, but if this is not done it is easy to spray the fluid into a minim measure,

and to determine how many emptyings of the ball are necessary before 10 minims have been sprayed. Supposing this number to be forty, the spray delivers 10/40, or one quarter of a minim, with each closure of the ball. If in this case a 10 per cent. solution of cocaine is used, 1/40th of a grain of cocaine would be sprayed with each emptying of the ball.

In certain cases of acute sinusitis there is no pain because the osteum is open. Because these cases are afebrile and painless they are very often missed. The treatment is the same in the early stages; the cocaine generally effects considerable improvement. If the fever and the secretion from the nose continue, and the secretion is for the most part post-nasal, the sinuses can be washed out, or in certain cases filled with liquid paraffin by use of vacuum. The latter is a new method, and under trial in acute cases.

In certain of these cases the fever is maintained, and when this is so it is generally advisable to administer either large quantities of fruit juice or large quantities of sodium bicarbonate, for often when the urine becomes alkaline the temperature falls almost as if by crisis, and inflammation in the nose resolves. In other cases alkalinisation does not have this effect, but it does no harm.

Subacute Cases.—Many subacute cases of nasal catarrh are very trivial, and are cured spontaneously. There are a few in which treatment is required because the cases have considerable importance. This importance arises by reason of the fact that certain forms of inflammation render the mucous membrane capable of toxic absorption to an unusually high degree, and filter-passing micro-organisms may, in certain rare cases, find their way through the nasal mucous membrane. It is, for example, believed that this is the source of infection in certain cases of encephalitis lethargica.

If in a case of subacute catarrh, symptoms such as diplopia, mental unrest, or failure of mental concentration are observed, it may be well to investigate the sinuses. It must be borne in mind, however, that there are many other causes for such symptoms, and many other portals of infection, and that catarrh is very common.

In the rare cases in which the nasal inflammation is the cause of such grave symptoms, lavage of the sinuses with liquid paraffin sometimes works wonders, for the liquid paraffin tends to prevent toxic absorption by covering the surface of the mucous membrane

with a layer of oil. In certain cases, even though a nasal sinus may be associated with these symptoms, the lavage of the sinuses with paraffin produces little alteration in the symptoms, because the micro-organisms have found their way into the cells of the mucosa.

In a few cases of subacute catarrh we meet with tonsillitis, which is secondary to the nasal inflammation. In such a case the posterior portion of the tonsil is more enlarged than the anterior, and the posterior pillar of the tonsil is much inflamed. In such a case the tonsils should not be enucleated at once, as the operation may debase the power of resistance in the sinuses, and lead to an increase in the activity of the infection.

In certain cases of subacute catarrh, particularly when a mild grade of sinusitis is preventing the recovery from a cold, it may be very helpful to try and empty the sinuses by a spray of liquid paraffin, which is actuated by an electric pump. This causes a strong blast of air and paraffin to pass over the opening of the sinus, which causes a vacuum within it in exactly the same manner as the escaping steam causes a vacuum in the inner chamber of the apparatus used for sterilising dressings. The head is placed in such a position that the partial vacuum will cause the pus to be emptied from the sinus. Sometimes this method works, and sometimes it is useless. If cases of sinusitis fail to clear up completely they should not be left alone, but should be washed out, preferably with liquid paraffin, in order to avoid the damage which otherwise will occur to the mucous membrane in the sinus.

Chronic Catarrh.—Many cases of chronic catarrh are trivial; others, by reason of the excess of the secretion, or its taste or odour, call for treatment, while others are more serious in that toxic absorption causes rheumatism, skin disease or similar remote effects. Among these latter there is an important group in which the patient gradually loses mental alertness and efficiency to an extent which is not infrequently prejudicial to his business. The treatment must be designed to fit the case. Thus in a mild catarrh which is not very excessive, and in which the discharge is odourless and tasteless, some simple palliative measures are all that are desired, and generally will bring about a cure.

Of these methods of treatment, a simple nasal douche, which is sniffed from a tumbler up the nose and blown from thence into a basin, is often very helpful. This does not, however, reach the interior of the sinuses, and if a sinusitis complicates the inflamma-

tion in the nasal airway, it is desirable to fill the sinuses with oil by the method introduced by Prøetz, and called displacement. If the paraffin is mixed with an oil opaque to X-rays, such as lipiodol, its entry can be verified upon the skiagrams. This gives us the information that the osteum of the sinus is open. If we can fill a sinus in this way, oil will be emptied from it by the action of the cilia, and with it the excess of mucus. If the sinus fills again, it probably should be washed out.

There are certain newer methods of displacement which are worth a trial. The X-ray photograph, after displacement, may show that one sinus has not filled, and this then may be washed out instrumentally if the discharge continues. In general, however, there is little need to wash out sinuses instrumentally in this mild group of inflammation, as they clear up very well by simple measures. In cases in which the discharge has an offensive taste or smell, or is so excessive as to cause considerable trouble to the patient, it is generally wise to examine the mucus in order to discover the type of cells, and whether free micro-organisms are present in these cells or in the mucus. If free micro-organisms are present it is probable that we shall not operate until other measures have been tried. If there are no free micro-organisms, then the patient must decide whether he will be treated by operative or by palliative measures.

In this class of case the results are obtained more quickly by operative than by palliative measures, and they are generally more sure; but if the patient is content to come week by week for palliative treatment, it will, in cases in which the discharge diminishes progressively, generally effect a cure. Such palliative measures as displacement frequently give good results, but they are tentative, and classified as a test treatment. Frequent washings out of the sinuses by instrumental means can be employed if the patient will permit them, but generally patients want some less disagreeable method, and simple displacement followed by nasal douching at home is generally the first choice, and if this fails, an operation is considered.

Before an operation is performed an X-ray examination of the sinuses should be carried out. If the X-ray shows thickening of the mucous membrane in all the sinuses, it is necessary to consider whether the case is one of allergy or whether the mucous membrane has been thickened as a result of inflammation. This is a very

important decision, because allergic cases withstand operation extremely badly. When the sinus mucous membrane is thickened as a result of inflammation, the master sinus, that is to say the one which causes the initial inflammation which has spread to all the other sinuses, shows a greater thickening of its mucous membrane than the others. If the mucous membrane is thickened equally throughout the sinuses, the case is generally one of allergy, and vaccines should be tried. These should be given by the intradermic route, and the degree of reaction of the skin controls the doses, which must be extremely small. In cases in which the thickened mucous membrane is confined to the antrum, and the discharge has not lessened as a result of a reasonable period of treatment, the antrum should be opened by Caldwell-Luc approach, and the mucous membrane carefully removed.

If the frontal sinus shows thickening of the lining mucous membrane, no operation should be carried out unless there has been pain which has not yielded to displacement; but if displacement has failed, the sinus can be catheterised or opened. The catheter will generally cause a remission of the symptoms, but will not cure in any but the mildest cases; but it is advisable to give test treatment in these cases before an operation is decided on.

If the inflammation is centred in one sphenoid, or one posterior ethmoid, the lavage can be easily accomplished through the normal osteum, but the opening of the sinus can only be effected by removal of the middle turbinate. If this is diseased, and the lining mucous membrane of the sinuses is seriously affected, operation may give the best results. In the majority of cases instrumental lavage of these sinuses is all that is required, and if there is recurrence it can be easily repeated.

In certain cases, the hypertrophy of the mucous membrane which has been produced by the inflammation leads to obstruction of the nasal airway. This obstruction is frequently increased in degree by reason of deflection of the septum. If an examination of the history shows that the nasal airway previous to the inflammation was comfortable, it is probable that removal of the focus of infection will restore the airway. If, in spite of treatment, the airway remains obstructed, the question of straightening of the septum and diminution of the hypertrophied turbinates will have to be considered, but this measure is generally inadvisable as a first procedure.

The only condition in which an operation has invariably to be carried out is that in which the mucous membrane has undergone polypoid degeneration, and when this is the case the altered mucous membrane around the base of the polypi must be removed. In addition, it may be said that the trend of opinion to-day is in favour of conservatism in nasal surgery, and that operation is reserved for those cases which have resisted the various forms of test treatment that are now in vogue.

CHAPTER XXXIV.

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

BY

CECIL P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S.E.

Senior Surgeon, King's College Hospital; Surgeon to the Belgrave Hospital for Children; Hunterian Professor, Royal College of Surgeons of England.

THE term congenital hypertrophic stenosis as applied to this well-known condition of the pylorus is far from satisfactory, but up to the present time no better one has been evolved. To state that no stenosis exists, and that the condition is simply due to pyloric incoördination, is simply begging the question. The reasons for still adhering to the term congenital are that the condition has been found at birth and in fœtuses at the eighth month of intra-uterine life. In all probability there are both ante-natal and post-natal factors which may come into play in causing the condition. The ante-natal causes being hyperplasia or spasm due to muscular incoördination, while the post-natal causes may be spasm or achasia. One of the outstanding features in the ætiology is the preponderance of boy babies, about ten to one. Further, over 50 per cent. of the cases occur in first babies.

The muscular hypertrophy extends from the pyloric veins on to the stomach as far as the incisura angularis. The hypertrophy does not affect the duodenum, which is normal in every respect; consequently there is a very sharp transition from the hypertrophic pylorus to the duodenum. This sudden transition must be kept in mind, as it is a dangerous area when division of the circular muscle coat is undertaken for treatment of the condition. The hypertrophic pylorus presents a firm swelling rather paler in colour than the remainder of the stomach wall, and its peritoneal covering is rendered very tense. On section the hypertrophied muscle is seen to be greyish-white and almost avascular (*see* Fig. 1). The

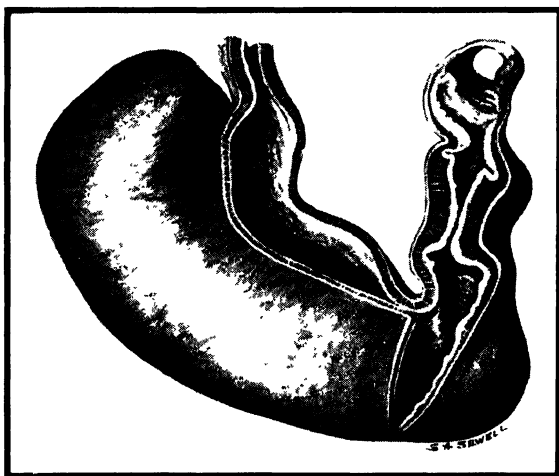


FIG. 1.—Specimen of congenital hypertrophic stenosis of the pylorus.
The great thickening of the musculature at the pylorus can be seen.
(From the Royal College of Surgeons Museum.)

‘CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS,’ By Cecil P. G
Wakeley, D.Sc., F.R.C.S., F.R.S.E.

submucous coat is thicker than normal, while the mucous membrane is often seen to be invaginated into the lumen, completely obliterating it. The stomach itself is markedly dilated, and shows signs of catarrhal gastritis.

The symptomatology of the condition is definite and characteristic, and can be briefly summarised as : projectile vomiting which is never bile-stained ; visible peristalsis ; a palpable tumour ; and obstinate constipation. The vomiting comes on gradually in a previously healthy baby, and soon becomes so explosive that an infant of six weeks may project its vomit for several feet. It is a curious fact that the infant is always hungry, and will immediately take a feed after a severe vomiting attack. The visible peristalsis can best be seen immediately after the child has been fed. The stomach outline appears as a rounded swelling just beneath the left costal margin. The swelling slowly becomes more distinct and more rounded in outline, until it assumes the size of a golf ball. When the swelling has become quite distinct it slowly migrates across the abdomen from right to left and then fades away. After a few seconds a similar wave of gastric peristalsis is seen. The best time to try and palpate the thickened pylorus is at the end of a peristaltic wave, as during the peristaltic contraction the pylorus is drawn downwards and to the left. At birth, and for some time after birth, the pylorus lies well under the inferior surface of the liver, which is relatively very large in the infant. The pylorus has quite a characteristic feel: its size and shape may be likened to a filbert nut ; it should be palpated with the tips of the fingers. It is useless to try and palpate the pylorus when the infant is crying, as the liver is forced downwards, and the abdominal muscles are so contracted that an examination is futile. For this reason it is necessary that the examination should be carried out when the infant is having a feed. Constipation is a very marked feature, and is due to the vomiting.

With such clear signs and symptoms the diagnosis should not be difficult, yet many cases are overlooked for want of proper examination, the medical practitioner concluding that the condition may be relieved by a change of diet.

The treatment for this condition should be definite, as in the past the mortality rate has been high because medical treatment has been tried first, and when this failed, surgical intervention was advocated as a last resource. Although Siegfried Wolff, of Eisenach,

has published some excellent statistics of his cases which have been treated medically, yet in this country no such good results have been forthcoming. The treatment to-day can be divided into three distinct parts: (1) Pre-operative; (2) operation; (3) post-operative. Neglect of any detail in any one part may be a factor in increasing the mortality rate.

The pre-operative treatment consists in giving the dehydrated infant as much fluid as its tissues will stand. No operation should be performed until subcutaneous saline has been administered. For an infant of six weeks, three ounces of normal saline to which 5 per cent. glucose has been added is given subcutaneously into each pectoral region and into each thigh. The solution should be allowed to percolate into the subcutaneous tissues very slowly. The infusion should be started about twelve hours prior to the time of operation.

An hour before the operation gastric lavage should be carried out, warm water being used for the purpose. It must be stressed here that on no account should sodium bicarbonate be used, as these infants are always in a condition of alkalosis, which is naturally increased should sodium bicarbonate be used. The operation which is advocated by all surgeons to-day is a pyloromyotomy, and of all operations devised Rammstedt's is by far the best, and is the one described below. After the gastric lavage has been performed, the infant's arms and legs are wrapped in cotton wool prior to its removal to the operation theatre.

Local anæsthesia is the method of choice, and I have used nothing else in my last seventy cases. Gas and oxygen anæsthesia is quite good if administered by a skilled anæsthetist, who does not allow any ether to be inhaled by the infant. However, skilled anæsthetists are not always to be found in country districts. On one occasion I performed Rammstedt's operation on a moribund infant for Dr. Still; no anæsthetic was used at all, and the infant recovered.

In the operation theatre the baby is placed on its back. One nurse holds the legs while another attends to the arms and gives the infant a comforter, which is frequently dipped in port wine, to suck. This prevents crying, and has a very soothing effect on the patient. The skin of the abdomen is washed with surgical spirit, and towels are placed in proper position. Some one per cent. novocain is injected by means of a hypodermic syringe under

the skin in the region of the right rectus muscle. By this means a wheal extending from the level of the umbilicus to the xiphisternum may be raised. Leaving the needle *in situ*, the syringe should be disconnected, refilled, and with a little further pressure the needle pushed into the rectus in order to infiltrate that muscle. The sub-costal angle is to be injected on both sides, as there is some overlap in the nerve supply of the skin in this region.

The actual operation is started about five minutes after the local anæsthetic has been given. A right supra-umbilical paramedian incision is used, and the rectus muscle is retracted outwards. On cutting through the posterior sheath of the rectus and peritoneum the liver is exposed, and presents in the wound. This organ, owing to its large size in the infant, prevents any of the intestines from herniating from the opening. By means of a pair of non-toothed dissecting forceps the liver is displaced upwards until its inferior border is raised above the lower limit of the incision when the stomach comes into view. The stomach is always dilated, and is to be gradually drawn out of the incision until the pylorus appears. When the pylorus is delivered through the incision the infant may cry, this being the only part of the whole operation which seems to be definitely appreciated by the patient, and is probably due to traction exerted on the gastro-hepatic omentum. The pylorus is so rotated that its upper surface looks directly forwards. An incision is made through the peritoneal coat and into the muscle coat. Then by means of a blunt-pointed probe the rest of the circular muscle fibres are divided, when the mucous membrane of the stomach will be seen to bulge into the wound. Very often the hypertrophy passes well on to the body of the stomach, and all the circular fibres in the hypertrophic region must be divided. Care must be taken not to perforate the gastric or duodenal mucosa. This accident is most likely to occur at the dangerous area, which is situated just distal to the pylorus, where there is a sharp transition from the thickened hypertrophied pylorus to the thin duodenal wall. Should a perforation occur it should be sutured immediately.

As a rule, hæmorrhage is very slight, as the hypertrophied muscle is somewhat avascular. Should it occur it must be stopped at once. The bleeding point can be underrun with a fine suture, or a small piece of rectus muscle can be excised and placed on the

bleeding area; in other cases a small tag of omentum may be sutured over the bleeding point.

Finally, the pylorus is replaced in the abdomen and the liver allowed to fall back into place, and thus provide a very efficient

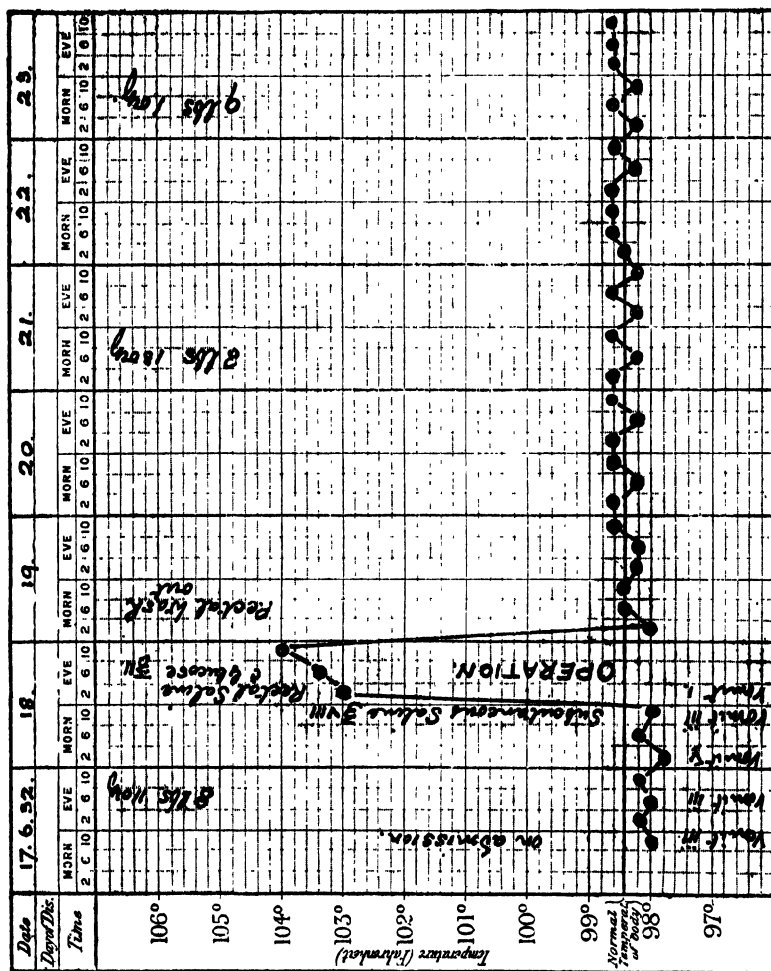


Fig. 2.—Temperature Chart of baby, John K., aged 6 weeks, before and after operation.

barrier in preventing any of the abdominal contents from getting into the wound. The peritoneum and posterior sheath of the rectus sheath are sutured with a continuous catgut suture. Should the infant suffer from shock to any marked extent, five to six ounces

of warm saline solution should be introduced into the peritoneal cavity prior to suturing. The anterior sheath of the rectus is sutured with catgut, and the skin with silkworm gut. A small collodion dressing is applied to the wound, and the dressing completed with a warm woollen binder.

Adrenalin should not be used in the local anæsthetic, as it seems to have no influence on the oozing, and when used frequently causes infection of the abdominal wound. The operation performed as thus described does not produce much shock and only takes a few minutes to perform.

The post-operative treatment is most important. The key-note is good nursing and small frequent feeds. The baby should have the exclusive attention of a nurse who should not be called upon to perform other duties. If the child is breast-fed, it should continue on the breast, as this is an important means whereby infection and post-operative diarrhoea may be prevented. After the child returns from the theatre three ounces of rectal saline are given. The first feed is given four hours after the operation, and consists of a drachm of 5 per cent. glucose, which is to be repeated after half-an-hour. After another half-hour two drachms of breast milk and six drachms of water are given at two-hourly periods, while in the interval an ounce of water is given. By this means the infant receives some fluid every hour. The breast milk is increased slowly, while the water is decreased to such extent that on the fourth day undiluted breast milk is given. If no breast milk is available, cow's milk, to which lactose is added, must be used ; it should first be peptonised for two and a-half hours.

As a rule the temperature often rises to 102° or 103° F. soon after the operation, but this calls for no alarm, and is due to the absorption of the large amount of subcutaneous saline given prior to the operation. The temperature falls as rapidly as it rises, and the day following the operation it should be normal (*see* Fig. 2). The infant gradually but surely gains weight, and can leave the nursing home or hospital ten days after the operation.

In the last 120 cases of congenital hypertrophic stenosis of the pylorus which have been operated upon by Rammstedt's operation at King's College Hospital and the Belgrave Hospital for Children, there have been five deaths only, or a mortality of 4.16 per cent. This small figure could be reduced to *nil* if cases were only submitted

to operation before the infant becomes emaciated. It must be said, however, that since the War there is a very definite improvement in this respect, and one confidently hopes that practitioners will seek early surgical aid in these cases. It is a real tragedy that the first-born son, a happy baby for six or eight weeks, should die of a condition which can be so easily cured by a comparatively simple operation.

CHAPTER XXXV.

THE DIAGNOSIS AND TREATMENT OF SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD

BY

T. KAY MACLACHLAN, M.A., M.B., F.R.F.P.S.

Assistant Physician, Glasgow Royal Infirmary ; Visiting Physician, Falkirk Royal Infirmary ; Consulting Neurologist, Glasgow Eye Infirmary ; Physician for Diseases of the Nervous System, Victoria Infirmary, Glasgow.

INTEREST has been renewed in this condition during the last six years following the great advance in treatment of Addison's (pernicious) anæmia by liver and its extracts, as initiated by Minot and Murphy ; more especially as the prognosis of this nervous disease, so often associated with pernicious anæmia, had formerly been extremely bad.

Subacute combined degeneration of the cord is a condition named from its histological picture. Degenerative changes are seen in the posterior and lateral columns of the cord, and in advanced cases practically all the white matter of the cord may show absence of myelin, while the brain also may show some shrinking due to demyelination, especially in the fronto-parietal regions. The disease tends to commence in the mid-dorsal segments of the cord where the blood supply is poorest ; it is thought to be of a degenerative rather than of an inflammatory or a vascular nature, and the striking feature is the absence of the neuroglial overgrowth which takes place in other conditions of the cord, whether they be of inflammatory or degenerative origin. The condition is frequently associated with Addison's anæmia, but may also occur with severe anæmia due to other causes, with lymphatic leucæmia, and with cachectic conditions.

Although subacute combined degeneration of the spinal cord is established as a definite clinical entity, yet combined disease

of the afferent and efferent systems of the cord may occur in association with several conditions, namely, disseminate sclerosis, Friedrich's ataxy, meningomyelitis, myelitis, lathyrism and pellagra, as well as in some syphilitic conditions where changes in the cord produced by interference with the vascular or lymphatic circulation may cause a similar picture.

One should not now be satisfied with a diagnosis of "spastic paraplegia" or "ataxic paraplegia," although these were included, till recently, in some text-books as diseases of the nervous system.

Subacute combined degeneration of the cord was formerly thought to be a late association of pernicious anæmia, and while it is true that symptoms referable to the nervous system, especially numbness and tingling in the extremities, can be found in the majority of patients suffering from pernicious anæmia, it must be remembered that similar symptoms may occur in any severe anæmia or cachetic state, and that one is not justified in diagnosing subacute combined degeneration on the presence of any nervous symptoms, even though there is no doubt clinically about the diagnosis of pernicious anæmia.

The criteria on which subacute combined degeneration has been diagnosed probably to some extent accounts for the extreme variations described by different observers in the response to treatment. It must, however, be remembered that subacute combined degeneration may be quite definitely present before there is any evidence clinically, or even hæmatologically, of pernicious anæmia, as ignorance of this point appears to be the reason why certain cases are so very far advanced before a diagnosis is made.

The diagnosis is not always straightforward, as the clinical picture varies, dependent on the relative affection of the posterior columns, pyramidal tracts, or peripheral nerves.

The earliest symptoms are usually paræsthesiæ, numbness and tingling in the lower limbs, and later the patient is aware of some clumsiness and weakness, and becomes less steady on the feet. Sensory examination shows some loss of vibration and joint sense, with perhaps some diminution in sensation to pressure and light touch. Less commonly there are pain, formication or a feeling of icy coldness, and lightning pains resembling those of tabes have been described. Later a girdle sensation, which may even be painful, develops, and paræsthesiæ also affect the hands.

It is usual thereafter to get involvement of the motor system, with a history of the limbs becoming easily tired, and later of one or both being dragged and feeling stiff; and this, with exaggerated deep reflexes, a positive Romberg's sign, clonus and extensor plantar responses, led to the condition being formerly known from its clinical features as ataxic paraplegia.

When the paralysis becomes marked, dysuria is usually noted, while if untreated the paraplegia becomes complete, and the trunk and arms are also involved, although usually the shoulder and upper arm muscles are little affected. In the later stages wasting of the muscles with loss of electrical response occurs, and a flaccid paralysis with absent knee-jerks, but an extensor plantar response, is seen, and there is incontinence of rectum and bladder. Less frequently the picture is one of flaccid ataxy with early loss of reflex activity and evidence of posterior column involvement, but even in these flaccid cases an extensor plantar response is usually obtained.

In the typical cases the sensory loss commences at the periphery, and tends to be of a glove and stocking distribution, while when it extends upwards to reach the trunk its progress is usually limited by segmental lines. Trophic changes are common when the disease is well established, and bed sores are then difficult to prevent. Vasomotor disturbances at the periphery of limbs also occur, and œdema is occasionally seen.

In the advanced case, evidence of involvement of the cerebrum is not uncommon, being shown by confusion, drowsiness, and occasionally by delirium. Changes in the pupils and in their reactions also occur, nystagmus may be present, and optic atrophy is occasionally observed, while if the disease has reached the lower cervical region involvement of the sympathetic fibres may be noted.

There may be no evidence of Addison's anæmia, but this is exceptional, it being usual to find at least megalocytosis with a high colour index. The tongue often indicates an existing or previous glossitis, but even a history of a painful tongue or recurring digestive upsets are of value in helping to confirm one's diagnosis. For further confirmation, the fractional test-meal should never be omitted, especially if the blood examination is not helpful, as complete or relative achlorhydria is found in practically all cases.

Diagnosis.

If when first seen the patient presents the picture of an ataxic paraplegia with some loss of postural and vibratory sensation, various conditions have to be excluded. Tabes, as well as specific meningomyelitis, must be eliminated by a routine examination of the spinal fluid as well as of the blood.

In support of a diagnosis of disseminate sclerosis there would be a more rapid onset, and one would look for a history of diplopia, or of transient paresis, blindness, paræsthesiæ or sphincter involvement, while nystagmus, absent abdominal reflexes, intention tremor or other lesions suggesting scattered involvement of the nervous system would be of confirmatory value. A paretic or luetic colloidal gold reaction in the spinal fluid, in the absence of other evidence of syphilis, would be of further diagnostic help.

If the girdle sensation or root pains were striking in addition to paraplegia and sphincter trouble, new growth of the spinal cord or its coverings would have to be excluded. Evidence of segmental sensory loss with perhaps a level of hyperalgesia, as well as pain and limitation of movement of the vertebræ, should call for X-ray examination of the spine, and this, together with Queckenstedt's test and examination of the spinal fluid for xanthochromia and for dissociation between the amount of albumin and the cell count, should establish or exclude the possibility of tumour. Lipiodol radiography is occasionally helpful.

In elderly patients the occurrence of paresis and paræsthesiæ in the lower limbs, as a result of vascular degenerative changes interfering with the nutrition of the spinal cord, must be considered.

The age at onset, the family history, as well as nystagmus and skeletal deformities, help to distinguish Friedrich's ataxia from subacute combined degeneration, while residence abroad, a diet containing *Lathyrus sativus*, and the simultaneous occurrence of several cases, would suggest that lathyrism was the cause of the paraplegia.

Pellagra is uncommon in this country, but a history of a deficient dietary, and the characteristic lesions of the skin, as well as the affection of the nervous and alimentary systems and the tendency to exacerbations in the spring of the year, should establish the diagnosis. It must be remembered, however, that the disease may occasionally occur without the cutaneous lesions.

Treatment.

There has been considerable difference of opinion as to whether liver, which is usually of such signal value in Addison's anæmia, is of comparable value in subacute combined degeneration of the cord. Some writers have claimed that all signs and symptoms of cord disease may disappear, while others have found liver ineffective, not only in causing no material improvement, but even in staying the advance of the disease.

We have already noted that the picture of degeneration of the spinal cord may precede any outward clinical evidence of pernicious anæmia, and in these days when the anæmia can be so easily and quickly controlled by liver at its onset, the dose of liver necessary for the treatment of anæmia *per se* need not be large for long. The failures in liver treatment of subacute combined degeneration of the spinal cord can often be traced to the employment of only those doses of liver which have been found sufficient to produce blood regeneration.

To treat the neurological condition it is essential that the liver be pushed to a greater degree, and at the onset it should be given both in the form of one of the intramuscular injections as well as by mouth, and both fresh liver and the extract should be administered by the latter route. As in pernicious anæmia, the response is better if the patient is able to take the fresh liver uncooked, and the daily dietetic routine can be made easier by the employment of the various recipes for the preparation of liver which have been published by Florence Irwin.

In combined degeneration of the cord one cannot be satisfied with a definite reticulocytosis and an increasing red cell count as indicating that improvement in the nervous condition will simultaneously occur. It must be our object to waste no time in arresting the changes in the spinal cord, and it has been found that a red cell count of five million is the standard at which one expects improvement in the cord symptoms, and not until this figure has been obtained should intensive liver therapy be discontinued. Naturally, one cannot expect regeneration in long-standing cases where nerve fibres have been destroyed, since nerve tissue cannot regenerate, but many of the symptoms have only been caused by the pressure of the swollen myelin sheaths, and intensive liver therapy, even in cases only diagnosed when the condition is well established, can usually relieve some of the symp-

toms. There are now many preparations of liver extract for oral administration on the market, and the equivalent of half a pound of liver as well as the same amount of the fresh gland should be given daily.

Following Gänsslen's production of extracts, potent by intramuscular or intravenous injection, the proprietary preparations Campolan and Hepatex are now available, and at the onset the equivalent of 500 grams of fresh liver should be given daily by injection.

Formerly, in cases with marked anæmia not diagnosed until an advanced stage, blood transfusion was often required, as the patient's anorexia and enfeebled general condition made adequate liver therapy by mouth impossible. Now, even if the nature of the nervous disease is not recognised until the anæmia is marked, transfusion is seldom necessary, or even intravenous injection, as an ampoule of Campolan twice daily intramuscularly is usually sufficient to initiate a remission.

The rise in the cost of mammalian liver since its value in anæmia has become generally known, and the greater expense of the liver extracts, has made treatment a serious economic problem in the case of sufferers from subacute combined degeneration of the cord, where treatment must be pushed to a much greater extent than in pernicious anæmia. In this connection the work of Davidson, in Aberdeen, in the preparation of a fish-liver extract promises to be of great value, as this extract has been found to be much more potent and more rapid in action than mammalian liver, while the cost to the patient should be much less.

Ventriculin, or other preparation of hog's stomach, which has a similar effect to liver in promoting blood regeneration, may be used in place of a liver extract; but, as with the latter, it must be pushed to a greater extent than in treating anæmia alone, and 30 to 40 grams daily should be given. The same remarks, however, do not apply to brain, which has been found to be of some value in inducing blood regeneration. In treating pernicious anæmia, Ungley found that the potency of brain in producing increase of the reticulocytes and red cells is only a third that of liver, but as regard the effects on the nervous system, brain has been found by him to produce definite neurological improvement in doses which would have relatively little effect compared with liver on blood regeneration. Brain is most potent

given raw, and can be administered in the form of a purée flavoured with tomato or orange juice. A dosage of about 480 grams daily should be employed.

More recently, Sargent, maintaining that some cases of subacute combined degeneration were not improved by liver therapy and that simple achlorhydric anæmia might be the cause of the persistence of neurological symptoms in certain cases, discarded liver therapy in favour of massive doses of iron, and from this treatment excellent neurological results were reported, and even hæmatological improvement took place in some patients when liver had been found to be ineffective. In these cases (simple achlorhydric anæmia) Bland's pill in doses of 150 grains daily may be used, and such an amount is usually well tolerated, even for the usual period of two or three months, before a reduction is made to a maintenance dose of 30-40 grains daily. It has been suggested that pernicious anæmia and subacute combined degeneration of the spinal cord are deficiency diseases, and Mellanby has experimentally produced a condition similar to subacute combined degeneration, even with mental affection, by diets deficient in vitamin A. He was able to relieve the neurological condition by incorporating vitamin A or carotene in the diet.

Strauss and Castle have shown that in pernicious anæmia there is absence of a normal "intrinsic factor" in the gastric juice. Normally this factor acts on protein in the diet to produce a hæmopoietic hormone, and the latter reacts with an essential "extrinsic factor," believed to be vitamin B, to cause hæmopoiesis. By further experimental work they conclude that vitamin B₂ is the extrinsic factor necessary for hæmopoiesis, and that deficiency of vitamin B₁ is the cause of the neurological symptoms. These investigations have led to the employment of yeast, which contains vitamins B₁ and B₂, in the treatment of pernicious anæmia and subacute combined degeneration of the cord. For those unable to take ordinary yeast regularly, this difficulty has been overcome by giving marmite, which is much more palatable, and a teaspoonful of this preparation should be given daily in sandwiches or added to gravy, while as a further variation bemax can be employed. If the diet does not contain adequate vitamin A, then the latter can be supplied by giving avoleum, radiostoleum or adexolin.

More recently Morris and his assistants have shown that a

hæmatopoietic response can be produced in pernicious anæmia by the intramuscular injection of gastric juice. The anti-anæmic substance, "addisin," which is probably a hormone, is destroyed by heat, and the juice has to be concentrated by distillation *in vacuo*. Amounts of this concentrate equivalent to 300–500 c.c. of gastric juice, while shown to be potent in the treatment of the anæmia, have yet to have their value proved in subacute combined degeneration of the cord.

Apart from what may be considered the specific treatment of the disease, there are other therapeutic measures directed to conditions frequently associated, which, if ignored, lessen considerably the efficacy of the methods we have already mentioned. Fever is not uncommonly present during the course of Addison's anæmia, and it does not appear to inhibit the action of liver; but if it is actually caused by any inflammatory or septic process such as cholecystitis, cystitis, or bedsores, then one finds definite inhibition of the normal response of neurological symptoms to treatment.

As in all nervous diseases, it must first be ascertained whether the bladder is functioning normally, or whether there is a bacilluria or pyuria. Direct questions must be asked in this connection regarding any dysuria, and it is well, even if the patient appears to be passing urine freely, to pass a catheter and find if there is any residual urine, as retention and decomposition of such often initiates a cystitis. Urinary antiseptics or lavage may be necessary.

Bed sores are of much worse import, especially if associated with incontinence of urine and fæces, and if diagnosis and treatment have been delayed they often precipitate a fatal issue. Any focal sepsis, whether in skin, teeth, nasal sinuses or elsewhere, must receive treatment, and constipation must, of course, be controlled.

Some patients with subacute combined degeneration manifest evidence of hypothyroidism, with malar flush, dry scaly skin, deficient perspiration, increased sensitivity to cold, and lowered B.M.R., and much general improvement may follow the administration of adequate doses of thyroid. The gastric deficiencies should also be made good, and the following mixture should be taken in a tumblerful of water with the three main meals of the day:

R. Acid hydrochlor. dil.	dram 1–2
Glycerin pepsin	dram 1
Syr. limonis.	dram 1/2
Aq.	ad oz. 1/2

If there is indication of intestinal decomposition, the administration of charcoal or dimol after meals may be helpful, while abdominal massage, recumbency after meals, or a supporting belt should be advised if there is evidence of visceroptosis. Massage and passive movements are of value in spastic cases, or in those debilitated by long residence in bed, and re-educative exercises are of the greatest help to those patients who have improved to the extent of commencing to walk again, as well as for those whose hands had become clumsy for fine movements.

The outlook of patients suffering from subacute combined degeneration of the spinal cord, formerly so hopeless, has been greatly improved within the last few years, but diagnosis still tends to be made too late to obtain the full advantage of the therapeutic measures now available.

CHAPTER XXXVI.

CÆLIAC DISEASE

BY

STANLEY GRAHAM, M.D.

Visiting Physician, Royal Hospital for Sick Children, Glasgow.

CÆLIAC disease, or the "coeliac disorder," is a condition in which there is present a chronic disturbance of nutrition characterised by the passage of very large, pale, foul-smelling stools, associated with distension of the abdomen and an arrest of growth and development. The condition was first described by Gee in 1888, under the name of the coeliac affection, and about a decade later was re-discovered by Herter, who referred to it as intestinal infantilism. In the United States of America it is most frequently spoken of as chronic intestinal indigestion, although the original title of coeliac disease is becoming more and more universal.

The disorder has occasioned a large amount of investigation both from the clinical and biochemical aspects, and more recently it has been suggested on good grounds that coeliac disease and non-tropical sprue are probably identical conditions. As the disease is essentially a chronic one, it is inevitable that there should be some delay before the diagnosis is established, or perhaps even suspected. A wider recognition of the characteristics of the malady will do much to ensure earlier diagnosis, and it is reasonable to assume that the sooner appropriate treatment is instituted the sooner will the healing stage be reached and the more readily will the various complications be prevented.

Clinical Manifestations.

The most common age of onset is roughly during the second and third years of life, although not infrequently one obtains a history dating back to the second six months of life. Instances of the onset being delayed till the fifth or sixth year are also on record, and

presumably, if the condition is identical with non-tropical sprue, it may occur at any age.

No particular section of the community is exempt, and in the children of the well-to-do it is certainly seen as frequently as in the poorer classes. It is said to be slightly more common in girls than boys.

The most characteristic feature of the condition is the passage of the bulky, porridge-like, offensive stools. The frequency as a rule is not great, and there may even be periods of constipation, although the bulk of the stool when passed remains excessive. The onset may occasionally be sudden with diarrhoea and vomiting, and perhaps some fever, but more commonly it is so insidious that no alarm is caused until the condition has been present for some time. The child loses his appetite, the weight begins to fall fairly rapidly, and the characteristic stools make their appearance. The abdomen becomes large and tympanitic, due to the gaseous distension of the atonic bowel wall. Mentally, the patient becomes ill-tempered, and nothing pleases him. From being a healthy placid infant he gradually becomes a miserable wasted child who tries the patience of mother and doctor alike. As this picture unfolds itself, the anorexia often becomes a most troublesome feature. The wasting becomes extreme, the skin, particularly on the inside of the thighs, hanging in loose folds. Miller¹ has called attention to the flattened buttocks as being a diagnostic feature. The muscles are hypotonic, and in all cases, even of moderate severity, the child "goes off his feet." The skin becomes sallow and at times slightly pigmented. An anæmia of the chlorotic type not infrequently makes its appearance. As the months go by the failure of growth and development becomes more and more apparent, until finally the picture of infantilism described by Herter becomes only too obvious. At this stage the picture presented is such that a superficial knowledge of the condition will lead to a ready diagnosis.

Apart from the anæmia which has been mentioned, and the frequent occurrence of the various infections, one other serious complication must be considered, namely, the development of "coeliac rickets."² As rickets can only make its appearance during growth, it is not so much during the active stage of the disease as during the periods of healing, either abortive or real, when growth is taking place, that this complication should be watched for. During the active stage severe osteoporosis develops owing to the

absence of sufficient lime and phosphorus. Tetany may also occur, and with the same metabolic disturbance as in infantile tetany associated with rickets.

Biochemical Manifestations.

The essential disturbance of metabolism in cases of coeliac disease is an inability of the gut to absorb fat in the usual manner. The absorption of carbohydrate is also considerably interfered with, as is also that of the minerals; but the consensus of opinion at the present time is that these two are secondary phenomena.

Fat.—Obvious evidence of the poor absorption of fat is to be obtained in the first instance by the history of the bulky stools due to the large amount of fat which they contain. The fat-splitting properties of the intestinal juices are unimpaired, and the condition can thereby be readily distinguished from pancreatic steatorrhœa, where the excess of neutral fat is so obvious. Although the stool may contain a large amount of fat, it does not necessarily follow that the percentage is increased. The amount of fat in the stool, other things being equal, obviously depends mainly on the fat intake, and while it may be true that the majority of cases of coeliac disease show a high faecal fat content, such a finding is not conclusive evidence of the presence of coeliac disease. The greater the intake of fat the higher the percentage in the faeces will be. In order to prove a diminished absorption (and hence retention) of fat, a balance experiment must be done. That is, the intake and output of fat over a given period, usually one of seven days, must be determined, and the percentage retention calculated. In coeliac disease, the percentage retention of fat is always diminished. It should be mentioned that in certain examples of tuberculous peritonitis where there is lacteal obstruction similar findings have been recorded.

Carbohydrate.—The defect in the absorption of carbohydrate can be more easily determined than that of fat. The blood sugar curve after the ingestion of glucose is "flat," or shows a rise of not more than 15 or 20 per cent. This is, however, not pathognomonic, as certain cases of simple diarrhœa during the acute stage may show a similar curve. Conversely, certain cases of coeliac disease during periods of remission may show a curve that is within the limits of normal. Nevertheless, in conjunction with the history and the appearance of the patient, a blood-sugar

curve will usually suffice to establish the diagnosis beyond all reasonable doubt.

Minerals.—There is no doubt that the diminution in the retention of calcium and phosphorus can be a serious feature. Retentions even lower than those obtained in infantile rickets are often found, and in this lies the explanation of the development of the severe cœliac rickets, especially during the healing stage. As previously mentioned, during the acute stage the diminution will result in an osteoporosis. The retention of these two substances can, however, be increased to normal by the use of some efficient non-oily preparation of vitamin D.

Protein.—Protein absorption is interfered with to a slight extent as well. Early in the disease large amounts of protein may be found in the stools, but it seems to be a fact that the simpler proteins, such as the casein of cow's milk, are absorbed well. On this assumption is the present-day treatment of the condition based.

Diagnosis.

In the early stages the condition may be readily mistaken for tuberculous peritonitis, and particularly is this so in those examples of the condition where the stools are not frequent and the attention of the mother is concentrated on the other manifestations of ill-health. A careful history, the examination of the abdomen for the presence of enlarged glands, free fluid, or rolled-up omentum, combined with, if required, the use of the tuberculin test, and a blood sugar curve, will enable one to differentiate the two conditions. Occasionally the condition is looked upon merely as malnutrition with rickets and "catarrh of the bowel." A knowledge of the nature and course of cœliac disease should prevent such an error.

Other conditions associated with infantilism are not likely to be confused with cœliac disease, and steatorrhœa from other causes can be ruled out by an examination of the stools. True pancreatic steatorrhœa, a very rare condition in children and occasionally congenital, will show the oily stool due to the high percentage of neutral fat present.

Ætiology and Pathology.

Practically nothing is known regarding the cause of this condition. Some maintain that there is an organic lesion present, but as far as has been shown there is no constant pathological picture.

Post-mortem material is limited, as the mortality of the condition is not great, and when death occurs there is usually some acute infection such as enteritis or broncho-pneumonia present to complicate the picture. MacRae and Morris³ have suggested that the fault lies in some physico-chemical abnormality of the intestinal contents.

Treatment.

The treatment is based on the assumption that the utilisation of fat and carbohydrate is defective, and that of protein comparatively normal. The patient is, therefore, given a high protein diet, the less complex proteins being used in the beginning. The following basic diet has been found to be a satisfactory one to start off with : protein milk or skimmed milk, 8 to 10 ounces four-hourly, five times daily ; curds from a pint of milk ; two bananas.

Protein milk (eiweiss milch) is troublesome to make, but can be obtained as a powder. Skimmed milk is probably as satisfactory, and can be obtained fresh from the dairy or as a powder. It is probably not wise to allow the skimming to be done at home. The relative percentages of the food principles in the two milks are as follows :

		Fat	Carbo- hydrate	Protein
Protein milk ...	...	1.5	1.5	3.0
Skimmed milk	...	1.0	4.5	3.0

If there is much diarrhœa, and especially if the fluid loss is considerable, it is advisable to give only the milk for the first few days and allow as much water as the child will take. Trouble may be experienced in the beginning in getting the patient to take the protein milk or skimmed milk, anorexia often being extreme. By perseverance in refusing to give any other food, and, if necessary, by the use of the stomach tube, this will soon be overcome. The curds should be well washed with water, and this drained off to remove as much of the fat and sugar as possible. It is a peculiar but incontrovertible fact that the carbohydrate in banana is especially well tolerated by these children, and they are of the greatest value in the successful treatment of the condition. The bananas should not be used until they are slightly over-ripe, with the skin turning black and the pulp soft and readily mashed.

Once the patient is satisfactorily started on this diet one can expect the weight loss to cease, and some improvement in the general

condition to take place. He will become more contented, and the gaseous distension of the bowel will be less because of the absence of carbohydrate fermentation. The banana may be increased in amount if he complains of hunger, but any great increase apart from this must be strictly avoided. The initial period of dietary treatment must last for several months at least, as relapses are very prone to occur, and in the end time will have been saved by prolonging this period. The patient is as a rule confined to bed for the majority of the time. In most instances he is unable to be up and about in any case because of the hypotonicity present. Later, additions may be made to the diet, such as scraped-beef sandwiches, beef juice, plasmon, calves-foot jelly, and certain varieties of cheese. Still later, chicken and rabbit as protein, and corn-flour, "Force," and biscuits as starch, may be added cautiously. Fats in any quantity are best avoided for at least two years, but much depends on the individual reaction to the treatment.

During the whole period it is advisable to give some active preparation of vitamin D other than cod-liver oil, or those preparations in which the ergosterol is dissolved in oil, because of the possibility of the non-absorption of the fat. As such a diet tends to be scorbutic in nature, orange juice or some fresh fruit juice should be given twice weekly. Vitamin B can be supplied as marmite.

The blood should be examined periodically, and if anæmia develops, some form of iron should be given. For this, ferri et ammonium citrate, ten or twenty grains thrice daily, is the most satisfactory. The addition of copper to the iron, as has been recommended, is probably not necessary, although there is no contraindication. It may be given as the sulphate in doses of a two-hundredth to a fiftieth of a grain thrice daily, depending on the age.

Course and Prognosis.

This depends to a great extent on the intelligent co-operation of the parents or nurse. There is no doubt that the earlier the treatment is instituted and the more thoroughly it is carried out the fewer the relapses and the more quickly will a cure result. In the hospital class of patient much difficulty may be experienced in obtaining the whole-hearted co-operation of the parents, and in these children one finds that relapses are more frequent and, as might be expected, the various infections are more common. Even in the mildest case under the best conditions it may be

impossible to prevent a relapse, and it is the exceptional case that goes through the whole course of the disease without some set-back. The duration of the dietary treatment is probably never less than two years, and in the majority of cases much longer, periods of five and six years being not uncommon in severe ones. If the condition has existed for a long time before coming under treatment, and there exists a well-marked infantilism, a cure may result in so far as the tolerance to fats is concerned, but some stunting of growth is inevitable. The condition has also been known to persist to adult life. The mortality is not high, but death occasionally occurs early in the course of the disease from an acute enteritis, and at all times the various infections take their toll.

REFERENCES.

- Miller, R.: Arch. Dis. Childh., Lond., 1927, II, 189.
- Parsons, L.: Arch. Dis. Childh., Lond., 1927, II, 198.
- MacRae, O., and Morris, N.: Arch. Dis. Childh., Lond., 1931, VI., 75.

CHAPTER XXXVII.

PINK DISEASE

BY

A. VICTOR NEALE, M.D., M.R.C.P.

Assistant Physician, Children's Hospital, Birmingham ; Assistant Physician,
Queen's Hospital, Birmingham.

DISEASES which are not uncommon in hospital practice may be distinctly uncommon in the average general practice. Pink disease is an example. Nevertheless, this disease is such a distinct clinical entity and has such characteristic signs as to render the diagnosis easy. The disease has received its appropriate and simple name from the fact that during the active or more acute stage the hands and feet possess a curious pink or red colour, not unlike the staining caused by red ink on the skin. This is essentially a disease of infancy or early childhood. In one hundred consecutive cases the maximum incidence occurred between six and twelve months of age. One case was seen in a girl aged seven years. On the other hand, seven cases were seen at the age of three months. All except four were healthy born, full-term babies. Of the one hundred cases, fifty-two were males and forty-eight females. In forty cases the disease affected the first and only baby in the family. There is no marked seasonal incidence, cases being seen all the year round, though more are seen in the second quarter. Clinically, the disease often commences in a baby who is in very good general nutrition, and careful inquiry will commonly establish no known deficient food factor ; in fact thirty-six cases occurred in well-nourished breast-feeding babies. There is rarely any evidence of nutritional or hygienic negligence preceding the onset. Undoubtedly pink disease may occur in babies who have first-class maternal care. By the time the patient is brought to a doctor, however, quite considerable wasting may have occurred and be an important sign in the general clinical picture.

Nothing conclusive can be said concerning the actual cause of this interesting disease. Some observers incline towards a nutritional deficiency factor, others believe the essential cause lies in an infective agent. No special micro-organism has yet been conclusively identified with the disease. Pink disease in its earliest phases nearly always shows a mild catarrhal or slightly suppurative upper respiratory tract disorder, with a variable degree of pyrexia. The question arises whether a specific virus is the real cause, or whether possibly the disease process is a special type of bodily reaction to a variable infective agent. The matter remains at present *sub judice*. If transmissible at all, a low degree of infectiousness would appear to be usual, since, although cases do occur in small epidemics, it is more usual for sporadic cases to appear. In this respect at least three instances of pink disease occurring in one of twins—each baby being cared for in exactly the same way in the same household—have been seen. There appears also to be no danger of spreading the disease in a hospital ward, since no secondary cases have been recognised under such conditions.

Signs of the Disease.—The onset is somewhat gradual. The usual story is that the baby was doing well until fairly recently, when signs of general irritability, restlessness, insomnia, anorexia, occasional vomiting, photophobia, limpness and various skin irritations and sweating appeared. The usual simple homely remedies or a change in the feeding have yielded no response, and the mother is disappointed. As a matter of fact, only few cases are seen by the medical attendant at this early stage, and nearly always by the time medical help is sought for the child has been ill for a period varying from four to more weeks. The gradual progression of the general misery, weakness, hypotonia, anorexia, sweat rashes, photophobia, constant crying, wasting, skin scratching, mild oral sepsis, and perhaps nasal discharge, and, above all, the progressive exhaustion of the patience of the mother, brings the doctor in touch with the patient. Most cases are recognisable at sight. It is a clinical picture quite distinctive. Degrees of intensity, however, are met with. Clinicians recognise that mild or probably short duration cases commonly occur wherein some or all the above signs are seen together, with a mild sweat or papular rash and slight pinkness of the hands and feet. In such mild cases wasting is negligible, and the total duration of all the illness may not exceed thirty days.



FIG. 1.—Posture of child in bed.



FIG. 2.—Posture of child in bed.



FIG. 3.—Eyes covered.—Photophobia.

In the average case, however, the bedside picture is usually complete: an unhappy child who has placed itself in such a position as to avoid the light. The posture is flexion, either laterally or quite often acute flexion at the hips in a sitting position, so that the child "closes itself up" with the head buried in the bed-clothes. Raising the head to the light reveals the photophobia, but the conjunctiva usually appears to be clear, and ophthalmoscopic examination reveals nothing unusual. The head is a distinct feature: the closed or semi-closed eyes, pink or red malar and nasal coloration with a surrounding pallor; the gums reddened and teeth often loose (they may be swallowed!); the tongue red and sore-like, or sometimes distinctly ulcerated, or even lacerated by the child's own restless mouth action; eyebrows thinned out, and the hair and scalp disturbed by trichotillomania and friction on the bed; a running nose and local excoriations completes the facial picture of misery and discomfort. The head droops on account of the weakened neck muscles. General hypotonia is evident, and a glance at the thighs and abdomen shows wasting of the muscles and looseness of the skin. The skin is moist or even markedly perspiring, and a variable degree of rash—sudaminal, macular or papular—is observed, principally on the trunk.

The hands and feet appear somewhat swollen, but true œdema is not recognisable. The pink, red or bluish-red colour (seen most markedly on the fingers usually) is striking. Peeling of the skin is common. In a small number of the more severe cases actual ulceration on the hands occurs, and is usually produced by gnawing. Secondary sepsis arising therein might be serious. Skin sensation may be diminished on objective test with pinprick, but the presence of cutaneous irritation, paræsthesia, etc., is evident from the scratching, restlessness and particular movements observed. Tendon reflex responses vary considerably: in the early stage increased responses occurring, but in the more serious advanced cases a diminution or absence may be elicited. Paresis is frequent, but paralysis does not occur. Evidence of visceral change or enlargement is conspicuously absent. The pulse rate is always increased (110–160), this being a very distinctive feature of the disease. Clinically no cardiac enlargement or change in the sounds occur. Blood pressure readings have often shown an elevation of the arterial tension. Pyrexia is absent; in fact nearly always (in the absence of intercurrent infections) the temperature is continuously

subnormal (97° F.) and remarkably "straight." Apart from occasional diarrhoea the stools are normal. A mild grade simple pyuria appears in some cases.

Conveniently the fully-developed disease may be epitomised by an alliteration of the words beginning with "P": peevishness, pinkness, pallor, perspiration, papules, peeling, pulse, posture, photophobia, paræsthesia, paresis, pyuria, etc. Individual cases vary considerably in their degree of severity. The mild character of the general clinical disturbance in some is specially interesting. The maximal general illness usually synchronises with the maximal degree of objective signs, such as the degree of sweating, rashes, photophobia, pinkness and hypotonia. In a few cases the objective signs are clearly present with apparently little general disturbance, and in such cases the total duration of illness is usually less.

Variations in the intensity of the physical debility may occur, and such may be accompanied by variable degrees of gastro-intestinal upset, severe anorexia, rapid fluctuations in weight, intensification of skin irritation, great restlessness and increase in pulse rate. The quickness of the pulse is usually proportional to the severity of the other signs. Fluctuations in the severity of the symptoms occur.

Cessation of fall in weight, diminishing tachycardia, lessening of the pinkness and swelling of the hands and feet, less marked photophobia, recovery of appetite, or return of the subnormal to a normal level temperature are indications that the maximal stage of the disease has passed. Certain phenomena, however, especially photophobia, muscular wasting and hypotonia, may be prolonged into a period when practically all other indications of the disease have abated. Wasting of the thigh muscles may take months to recover. The total duration of the disease in moderate or severe cases is remarkably protracted. This is one of the few severe prolonged illnesses of early life which recovers completely in high percentage. In most cases of average severity definite illness occurs for 100 days. Numerous instances of cases showing definite signs of 150 days have been seen. The average duration of 21 cases under the age of six months was 95 days, whereas that of 16 cases over the age of eighteen months was 160 days. Generally, the older the child at the onset the greater the probability of the disease being of longer duration.

Clinical Examples : (a) Having a Relatively Short Duration.

J. S.—Female, aged six months. Birth weight, 8 lb. ; breast fed. Had reached twice the birth weight in five months. Two weeks before admission, anorexia, and appeared distressed ; rubbing the eyes ; cutaneous itching ; insomnia ; photophobia. Condition on admission : well nourished ; red hands and feet ; nasal catarrh ; very irritable ; slight hypotonia ; sensation normal ; reflexes brisk ; urine normal ; pulse 130. Breast and cow's milk, sago pudding, soup, egg yolk, mashed potato ; ultra-violet light given. In six weeks considerable improvement, skin signs gone, pulse still 130. Net less of six ounces in weight. Taken home for a week, and readmitted with relapse of the photophobia, which gradually cleared up. Final weight 16½ lb. Pulse 120. In hospital forty days ; illness sixty days.

J. E.—Male, aged two years. Birth weight, 12 lb. Varied diet. Catarrhal infection six weeks before admission, and not well since ; became peevish and weak. Condition on admission well developed ; very irritable ; typical pink signs ; muscular hypotonia marked ; no photophobia ; marked perspiration. R.B.C. 4,200,000 ; W.B.C. 13,200. Urine normal ; pulse 150 ; weight 1 st. 12 lb. Treated with varied diet. Liq. arsenicalis m. ii t.d.s., fourteen days. Progressive improvement. In hospital thirty-three days ; total illness seventy-five days. On discharge, weight 2 st. 1 lb ; pulse 100.

E. H.—Male, aged six months. Birth weight, 9 lb. 4 oz. ; breast fed. Three weeks running eyes ; catarrhal nose ; early slight photophobia ; papular rash. Condition on admission : miserable and irritable ; characteristic pink signs ; great difficulty in feeding ; reflexes brisk ; afebrile ; pulse 140 ; weight 17 lb. 6 oz. Treatment : cow's milk, whey, ultra-violet light. Pulse dropped to 120 ; weight rose to 18 lb. In hospital forty-eight days ; total illness seventy days.

E. S.—Female, aged five months. Birth weight, 7 lb. Breast fed. Ill four weeks ; crying, irritable ; loss in weight ; transient rashes ; red hands and feet, which began to peel ; photophobia. Condition on admission : good development ; typical pink disease ; hypotonia ; very miserable ; photophobia definite ; pulse rate 140 ; weight 11 lb. 14 oz. ; apyrexial. Treatment : breast feeding, ultra-violet light, radiostol. Gradual disappearance of signs of

the disease. Pulse declined to 120; weight increased to 12 lb. 12 oz. In hospital twenty-eight days; total illness, sixty days.

O. S.—Female, aged five months. Birth weight, 9 lb. Ill fourteen days—irritable; occasional vomiting; continuous crying; early photophobia; soon developed red hands and feet; sweating and papular rash. Condition on admission: well nourished; very irritable; peevish; photophobia moderate; muscles flabby; pulse 150; weight 14 lb. 14 oz.; apyrexial; urine normal. Treatment: breast and cow's milk; vegetable soup; radiostol. Gradual improvement, photophobia being the last visible sign to disappear. Pulse gradually fell to 130. In hospital thirty-five days; total illness sixty days.

(b) Having a Relatively Long Duration.

J. H. J.—Male, aged one year four months. Breast fed for twelve months. Three months before admission started to pull hair out; went off his feet; hands and feet red and swollen; small ulcers on tongue. Condition on admission: typical features of pink disease; red peeling hands and feet; generalised papular rash; marked adductor wasting; severe hypotonia; R.B.C. 5,000,000, W.B.C. 11,500; weight 17 lb. 3 oz.; pulse 150; afebrile; subnormal temperature. Treatment: full varied diet; ultra-violet light. In hospital photophobia developed; hypotonia increased; after two months in hospital photophobia much less; able to stand up. Soon afterwards recrudescence of photophobia; rash; pinkness and sero-purulent nasal discharge. Four weeks later more agreeable; pinkness and photophobia still present. Taken home still hypotonic, some pinkness of hands and feet. Seen again one month later: knee jerk brisk; gaining weight; no analgesia; hair growing rapidly; much improved muscle tone. Readmitted to hospital ten weeks later with ulcers on tongue; pyorrhœic gums. Signs of pink disease abated. Weight 23 lb.; pulse 100. Illness before admission twelve weeks. In hospital 114 days; total duration of illness 200 days.

R. F.—Female, aged ten months. Birth weight, 9 lb. Cow's milk. Seven months before admission body came out in blotches; became miserable; restless; free perspiration; steadily lost weight. Symptoms continued without any improvement. Condition on admission: typical pink disease; marked photophobia; perspiration; hands and feet peeling; good gastric secretion; c.s.f.

normal. Blood—R.B.C. 4,000,000 ; Hb. 60 per cent. ; W.B.C. 8,000 ; pulse 135 ; weight 12 lb. 8 oz. Treatment : milk ; ultra-violet light ; ol. morrhual and iron. In hospital the photophobia and hypotonia became extremely marked ; rash gradually cleared ; gradually all signs abated ; pulse declined to 120 ; gained 3 lb. ; afebrile ; subnormal temperature all through. Ill twenty-eight weeks before admission ; in hospital 130 days ; total illness 340 days.

S. B.—Male, aged seven months. First symptom appeared aged three months—photophobia (taken to eye hospital) ; irritable skin, severe incessant scratching, and soon afterwards severe perspiration and red hands and feet. Condition on admission : well nourished ; very fretful and irritable ; marked photophobia ; typical posture ; obvious cutaneous irritability ; hands and feet deep-red colour ; active peeling ; mild conjunctivitis ; gross hypotonia ; tendon reflexes brisk ; urine normal ; pulse 140 ; weight 15 lb. Treatment : whole milk ; Almata ; rusks ; potato ; gravy ; soups ; custard ; cereal ; calcium lactate, gr. iv, t.d.s. Signs showed gradual abatement, but pinkness and severe perspiration persistent ; photophobia severe ; some intermittent exacerbation of signs and rash ; photophobia disappeared only very slowly ; finally discharged as cured. Ill sixteen weeks before admission, in hospital seventy-five days, total illness 200 days.

D. H.—Male, aged eleven months. Ill for three months. Perspiration ; miserable ; photophobia ; irritability ; coryza ; red hands and feet. Condition on admission : intensely miserable ; cutaneous hyperæsthesia ; typical pink coloration ; pulse 145 ; weight 17 lb. Treatment : full varied diet ; liver. Gradual progressive improvement ; pulse declined to 105 ; gained 3 lb. Before admission ill for twelve weeks ; in hospital eighty days ; total illness 170 days.

Recognition of the disease in an early phase, even where severe signs and symptoms already exist, is important, since institution of good nursing control will tend to shorten the duration of the disease and possibly avoid such troublesome phenomena as protracted photophobia, skin infections and wasting.

Intercurrent Phenomena.—During the active disease patients are very susceptible to various superadded infections. Mild skin infections are not uncommon, but a spreading sepsis leading to ulceration is dangerous. Serious ulcero-necrotic areas may appear

in the perineal or oral areas, or the hands. Acute lung œdema sometimes appears. Broncho-pneumonia is the cause of death in practically all fatal cases. Occasionally sudden unexplained (? cardiac) dissolution occurs during the height of the disease. Two cases of acute mastoiditis and one of acute intussusception during the pink disease have survived surgical treatment. It is interesting that a sharp intercurrent pyrexial attack may lead to rapid disappearance of the pink signs. This occurred in the two mastoid cases.

Some Cases of Special Interest.

A. P.—Male, aged three and a-half months. Birth weight, 10 lb. Cow's milk; progress good. Ill for five weeks before admission: fretful; restless; anorexia; insomnia; sweating; photophobia; blistered mouth; marked rashes of maculo-papular type all over the body; typical pink puffy fingers and toes; severe hypotonia. During six weeks' treatment in hospital, rapid clearance of signs, and became active and sitting up in bed, the pulse having fallen from 150 to 120. Lactic acid milk (*Streptococcus lacticus*) prescribed only. Interesting features were (a) severity of the signs in so young a baby; (b) rapid response to hospital supervision; (c) lactic acid milk used only as the diet; (d) a cousin had pink disease at the same time.

G. B.—Male, aged three years. Photophobia, insomnia, rashes, pink hands and feet, peeling, perspiration. Condition on admission: typical severe pink disease; tendon reflexes absent. Symptoms and signs intensified soon after admission. A series of blood-pressure readings showed repeated result of systolic pressure 125, diastolic pressure 95. Apyrexial for first five weeks in hospital; pulse 140. Suddenly developed acute mastoiditis with pyrexia to 104° F. Operation. Following this there was a rapid cessation of the previously marked signs of pink disease, and the pulse declined to 110. Temperature became subnormal, and the normal diurnal variation returned. Gained 4 lb. in weight. Discharged after being in hospital for 130 days cured. An instance of severe pink disease developing intercurrent acute pyrexia and mastoiditis, and the curious accelerated disappearance of signs of active pink disease immediately afterwards.

L. B.—Male, aged nine months. Breast fed; fat child; flabby muscles; restlessness and irritability. Typical pink signs in the

marked hypotonia; pulse 130; cerebro-spinal fluid normal. Admitted, and placed on a light varied diet; radiomalt; ultra-violet light; liver extract. Five weeks' in-patient supervision allowed very considerable improvement, and the baby returned home. Four weeks later readmission to hospital was necessary on account of pronounced photophobia and a recrudescence of the pinkness and other signs. Pulse had risen again to 130. Three weeks' further hospital supervision allowed rapid cessation of the signs, and cure was established, although the pulse rate remained up to 110.

Remarks.—Relapses are very uncommon, and a second attack of pink disease is unknown (in a series of 100 cases). This patient definitely relapsed clinically, and photophobia became severe; this might have been due to exposure to powerful sunlight (August).

Therapeutics.

In the absence of complications there is little doubt that pink disease tends towards spontaneous cure, providing an adequate food supply is received and good nursing applied. In view of the unknown factors in the ætiology there is no specific form of treatment yet available. The essential matter is: how can the active phase (which is probably a neuro-circulatory effect of an initial infection) be minimised in intensity and duration? Recognition of the disease in its early stages is important. Tachycardia, restlessness, perspiration, rashes and photophobia are important early phenomena. General points, such as a warm, well-ventilated, but shaded room; soft, non-irritating bedclothes, and pillows arranged in hammock-fashion; a cot is essential, or the child may writhe and fall out; leave the child to move about at will in its cot (it will find its own position of maximum comfort). A good nurse is essential. Care of the skin is very important. The arms may be "cuffed" to prevent scratching of the body and chewing of the hands. Irritation can be allayed by sponging with a weak alkaline fluid. A 5 per cent. menthol in vaseline allays the severe irritation of the hands and feet. Simple general sedatives are useful. Special care is required to prevent secondary infection of the skin, especially of the buttock, face and scalp, and oral hygiene needs close attention. Food of good wholesome type is of fundamental importance. Successful administration of such food to the affected infant or child is almost the most important

aspect of the whole problem. Breast feeding should be continued wherever possible, or whole cream milk instead. Varied dietary is required for an older baby, and should be persisted with. Many special additional materials have been used with benefit, such as mashed potato, egg yolk, dried yeast, soups, ol. morrhuae, minced liver (up to 1 oz. daily), radiostol, adexolin, calcium lactate, marmite, etc. Strychnine, arsenic, adrenalin, pituitrin, are of no benefit, but a few grains of ferri et ammon. cit. in the later stage are helpful. First class nursing and feeding (such as is obtained in a modern children's ward) undoubtedly allays much of the little patient's misery, hastens recovery and minimises the occurrence of dangerous sepsis. The use of ultra-violet irradiation is of definite value—two or three short exposures a week often acting sedatively, and it appears to keep up the patient's physique. Pyuria (usually *B. coli*) calls for the usual remedies. Teeth should be watched carefully, and any loose ones removed, or their inhalation may take place.

Convalescence is usually slow, and normal muscular power is not to be expected for several weeks after an otherwise clinical recovery. The pulse rate only very slowly reaches normal. Recrudescence of symptoms may occur if any negligence occurs in the convalescent period.

Ultimate Prognosis.—When one sees pink disease in "full bloom" the question immediately arises as to whether any permanent effects or sequelæ will ensue. The answer is, that in no case where the disease has been successfully weathered has any direct or indirect ultimate disturbance been discovered. This statement is based on a careful re-examination of about sixty children who had suffered from the disease two or more years previously. Even in the prolonged and clinically very severe instances the patients have made a complete and permanent recovery, and are now mentally and physically normal in every way. The present excellent physique is remarkable when seen in contrast with their photographs taken during their previous state of abject misery. Also electro-cardiographic tests show that the heart is normal. There is no residual abnormal blood pressure.

CHAPTER XXXVIII.

THE CLINICAL SIGNIFICANCE OF ERYTHEMA NODOSUM

BY

W. R. F. COLLIS, M.A., M.D., F.R.C.P., D.P.H.

Physician to the National Children's Hospital; Pædiatrician to the
Rotunda Hospital, Dublin.

ERYTHEMA nodosum, although fairly uncommon in general practice, occurs much more frequently than is often believed. Its ætiology until recently has been shrouded in mystery. Even the most modern text-books only supply the vaguest generalisations as to its cause. An understanding of its ætiology and its subsequent correct diagnosis by the practitioner is of the greatest importance, however, both as regards the theories of immunology and the practice of therapeutics. It is with regard to the latter point of view that this article has been written. Before attempting to outline the treatment in any given case of erythema nodosum, it is necessary to have a clear idea of (1) the disease syndrome involved, (2) the clinical significance of its ætiology.

Clinical Description.

The nodular eruption associated with the disease is its most characteristic sign, but it must not for this reason be considered a skin disease. (Erythema nodosum has all the signs of a general body reaction); it is preceded by an indefinite period of malaise, during which there is complaint of headache, lassitude, anorexia, and in some cases muscle and joint pains. Fever is usually present during this period. The time of the occurrence of the eruption is variable. The nodes usually appear at once, but sometimes do not come out until the second and third week of the fever. The lesions are characteristic, appearing on the skin as red, raised, indurated nodules of varying size and type. They may also occur on the arms, trunk and face.

The lesions soon fade, leaving a bruised discoloration, and the patient may recover within a week. The actual attack is seldom severe, and never fatal, and only requires rest in bed and palliative treatment. The importance of the disease lies in its ætiological relationship to tuberculosis and occasionally acute rheumatism, as will be demonstrated later.

Ætiology.

During the last fifty years considerable controversy has raged over the ætiology of the disease. In England, due to the work of Mackenzie in 1886, the condition has been held to be associated in some way with acute rheumatism, though the connection has never been clearly defined. This view has, however, become incorporated in the English text-books; indeed, almost every case of the disease admitted to hospital in England or Ireland is now labelled acute rheumatism by the practitioner. The rheumatic hypothesis rested on the observation (that certain cases of erythema nodosum followed acute tonsillitis in much the same way as rheumatic fever), that these cases were often complicated by severe polyarthritis, and that rheumatic heart lesions occasionally followed the attacks. The most ardent supporters of this view were unable to claim that the majority of cases of the disease presented these points, and all had to admit that certain patients presented no symptom of or connection with acute rheumatism. On the Continent a different view has been coming into prominence, due to the work of Ernberg, Wallgren, Pollack, etc., who have pointed out (the connection between erythema nodosum and primary tuberculous infection). They have shown that a high percentage of patients with erythema nodosum are positive to tuberculin tests, and that the hypersensitivity to tuberculin appears at the same time as the appearance of the eruption, which is associated with phlyctenulæ in 30 to 40 per cent. of patients, and radiological evidence of lung lesions in over 60 per cent., while gastric lavage will often reveal the presence of tubercle bacilli of the human type. They also observed that frank tuberculosis often follows erythema nodosum (as much as 50 per cent. in one series), and that it is usually preceded by a definite history of tuberculosis contact infection.

Possibly the most noteworthy of these observations was that made by Wallgren, who described the development of a fever in eighteen girls at a school; the fever was associated with erythema

nodosum in twelve cases, the whole epidemic occurring during one term. Close examination showed that all the patients were positive to tuberculin, thirteen showed definite lung lesions, and a case of open phthisis was found in the school. In spite of these facts, however, even the most ardent supporters of the tuberculous hypothesis were unable to explain all cases, some always being tuberculin-negative and apparently in no way connected with tuberculous infection. As it appeared that neither the rheumatic nor the tuberculous school could claim all cases, a third hypothesis was suggested by Lendon, Trousseau, Hutchison, and others. These physicians suggested that the disease was neither rheumatic nor tuberculous, but an acute specific exanthem, distinct from both.

Some years ago the present author took up the study of the disease at the Hospital for Sick Children, Great Ormond Street, London, and has since followed up the work in Dublin. A communication on the subject was published in the *Quar. Jour. Med.*, Jan., 1932, and the findings there set forth may be summarised briefly as follows :

Eight children with the disease were studied in detail, every modern method being used in their investigation. The cases fell into two groups. In the first group all the patients were positive to tuberculin, negative to streptococcal extract, showed lung lesions ; in three out of five tubercle bacilli were obtained by gastric

TABLE SHOWING TYPES OF ERYTHEMA NODOSUM.

			TUBERCULOUS	RHEUMATIC (STREPTOCOCCIC)
Family history	...	...	History of relative with active pulmonary tuberculosis	No family history of tuberculosis ; occasionally family history of rheumatic fever
Previous history	...	...	No previous history of tuberculosis or rheumatism	Sometimes past history of subacute or acute rheumatism
Present attack	Type of eruption	...	Tends to be small and diffuse	Tends to be large and confined to the legs
	Number of attacks	...	Usually occurs only once	Multiple attacks common
	Preceding Pharyngitis	...	Absent	Present
	Accompanying Polyarthrititis	...	Absent	Present
Skin Tests	Tuberculin	...	+++	Usually negative
	Streptococcal extract	...	Usually negative	+++
X-ray of chest	...	...	Shows primary tuberculous lesions	Normal
Sputum (gastric lavage)	...	...	Often positive for tubercle bacilli	Negative

lavage ; all gave a family history of tuberculous contact ; all were suffering from their first attack, and none gave a history of preceding sore throat or complained of joint pains during the attack. In the second group all were negative to tuberculin, positive to streptococcal extract, gave no history of contact, had no lung lesions, gastric lavage was negative, all had a history of preceding tonsillitis and accompanying joint pains.

These findings have been subsequently confirmed by the present author and others on a much larger series of cases, and it is now becoming recognised that erythema nodosum is a type of body reaction which may be brought about by more than one type of infection, provided the patient is in a state of hypersensitivity to the products of the germ in question. The most usual infections associated with the disease are tuberculosis and hæmolytic streptococcal infections, but it is believed that other infections may very rarely cause the disease syndrome.

In another communication it is hoped to publish the actual percentage figures of the different types from various centres. It may be stated here, however, that by far the largest number (over 70 per cent. in London and 90 per cent. in Dublin) belong to the tuberculous group. This is important in view of the common practice of calling all cases of the disease rheumatic in England and Ireland. Actually our attitude should be the exact opposite, and all cases should be considered tuberculous till proved otherwise.

Diagnosis into Type.

The foregoing table sets out the main differences between the two types, and if followed will make the diagnosis comparatively simple in most cases. Occasionally in a multi-sensitive case the diagnosis may be difficult, and a definite decision may be impossible, but the majority may be differentiated if this method is followed.

Treatment.

As already stated, the majority of cases of erythema nodosum belong to the tuberculous type. This means that they have had a recent primary tuberculous infection usually by the pulmonary route. It necessarily follows that there is a source of tuberculous infection in their immediate environment which should not be too difficult to detect, particularly in the case of children. Direct

questioning does not always elicit the necessary information, as the following case shows :

"A girl, aged ten years, was admitted to the ward suffering from erythema nodosum (and labelled, as usual, 'acute rheumatism') ; investigation showed that she was definitely of the tuberculous type, and human tubercle bacilli were obtained by gastric lavage. On questioning, the mother declared that the whole family was healthy, and no history of contact could be obtained. It was possible, however, to examine the whole family, and by this means it was discovered that a brother of seventeen years had an unsuspected phthisical cavity (and died later from generalised tuberculosis), and a small sister also had been infected. The latter had been losing weight for some time, was strongly positive to tuberculin, and X-ray showed a primary lung lesion, and lavage revealed human tubercle bacilli in the swallowed sputum."

This case illustrates the great importance of making a careful follow-up of all cases of the tuberculous type of erythema nodosum, so as to find out and eliminate the source of infection.

It is impossible to enter here into a discussion on the ætiology and prognosis of primary pulmonary tuberculosis. Suffice it to say that the lesion in this primary state is altogether different from that of phthisis, and that the prognosis is good provided the correct treatment is possible.

These patients do not require the sanatorium type of treatment given for phthisis. They require merely ample rest, good diet, open air, and protection from intercurrent infections, in other words, prolonged convalescent home treatment. If they are accorded this form of therapy most will recover within six months, although all should be carefully supervised for several years after the attack.

The rheumatic or streptococcal type comprise the majority of the remaining cases. These patients are found to be highly sensitive to hæmolytic streptococcal extract, and tend to have recurring attacks every time they get a hæmolytic streptococcal infection.

Tonsillectomy does not appear to prevent subsequent attacks, but if infected tonsils are present they should be removed. Until some specific method of desensitisation has been elaborated, treatment of these cases can only be palliative. Vaccine treatment may be tried, and all sources of infection should be excluded as far

as possible. The actual attack should be controlled by large doses of aspirin or pyramidon (gr. x, three times a day, to a child of ten years). Those cases which do not fall into either the tuberculous or rheumatic type are so rare as to be of academic interest only.

Conclusion.

Erythema nodosum is a type of hyper-reactive tissue reaction to the breakdown products of different organisms. It should be remembered, however, that much the commonest cause is the tubercle bacillus, and the doctor's attitude should be to regard every case as tuberculous until proved otherwise.

CHAPTER XXXIX.

TREATMENT OF ANÆMIA IN INFANCY

BY

HUGH W. JOSEPHS

From the Harriet Lane Home of the Johns Hopkins Hospital and the Department of Pediatrics, Johns Hopkins University, Baltimore, Maryland, U.S.A.

Physiological Anæmia.

PRACTICALLY all infants are born with hæmoglobin value of 100 per cent. or above. During the first month or six weeks the hæmoglobin falls to 70 per cent. or below. It used to be considered that 65 or 70 per cent. was a normal hæmoglobin value throughout infancy, and the term "physiological anæmia" was applied to this relatively low hæmoglobin figure. Lately, however, especially since the work of Helen McKay, it has come to be realised that a low hæmoglobin throughout infancy is not inevitable, and the term "physiological anæmia" is now applied only to the early period of a month or six weeks, during which the fall in hæmoglobin must be regarded as part of the adjustment to extra-uterine life. Except for this early period of adjustment the tendency of the hæmoglobin to remain at relatively low values depends on factors which are to a large extent capable of being influenced by treatment. The most common of these factors are: (1) prematurity; (2) lack of iron; (3) infections.

Anæmia of Prematurity.

The period of unavoidable or "physiological" fall of hæmoglobin lasts longer in the premature than in the full-term infant, and the hæmoglobin tends to fall to a lower level. The duration of this early period depends in general on the degree of prematurity, but seldom lasts longer than ten weeks, while the hæmoglobin rarely falls below 45 or 50 per cent. The hæmoglobin of the prema-

ture, unlike that of the full-term infant, shows little tendency to rise merely under the influence of good hygienic conditions, and it is necessary to use treatment directed specifically to raising the hæmoglobin. The simplest and most effective form of treatment is the administration of iron, which at this time acts as a stimulator of hæmatopoietic tissue. Lack of iron as a specific substance in the formation of hæmoglobin plays no part in the anæmia of prematurity before the end of the third month of life.

Lack of Iron.

The amount of iron in milk is insufficient to supply more than a small proportion of the iron needed for hæmoglobin formation during infancy. If it were not for the presence of iron stores at birth a deficiency of iron would probably develop in all babies before the time that iron-containing foods could be safely given. Judging from figures in the literature, the amount of iron with which the infant is born is at best sufficient to maintain the hæmoglobin at normal levels up to about the ninth month, but probably in the majority of cases not beyond the sixth or seventh month. Since it is not possible in the individual case to determine when the iron stores are becoming exhausted, it is best to be sure that every child is supplied with iron-containing foods before the sixth month. If by this time the hæmoglobin has shown a tendency to remain below 75 per cent., as may happen in cases in which the infant has had poor hygienic surroundings, or has been frequently exposed to infection, it would be well to supplement the food iron with some medicinal iron preparation.

Although in the individual case we have no means of determining the amount of available iron, we do know that in premature babies, twins and others who are underweight at birth, the iron stores are likely to be inadequate for the maintenance of hæmoglobin, so that in these cases, especially in the premature, we encounter iron deficiency earlier, in some even as early as the end of the third month. According to the work of Strauss, it is probable that babies born of mothers who have suffered from hypochromic anæmia during pregnancy will have somewhat deficient stores of iron. In all these cases, where one has reason to believe that the iron stores are inadequate, medicinal iron should be given in large doses, beginning in the second or third month, and should be continued until the hæmoglobin rises above 80 per cent. and remains above this level.

Infections.

The later a condition of iron deficiency occurs the less its effect on the hæmoglobin percentage, so that ordinarily deficiency of iron by itself would not tend to produce any great degree of anæmia unless unduly prolonged, except in the premature and others in whom iron deficiency develops early. Infections, on the other hand, not only may cause a rapid development of anæmia, but the effect of an infection may be prolonged far into convalescence. This is due primarily to two facts: (1) During a severe infection there is likely to be considerable loss of iron from the body, and if the infection is at all prolonged the stores may be materially depleted. (2) Following an infection in some cases the appetite may be so reduced that for a long time nothing but milk will be taken, with the result that an anæmia starting with the infection may be prolonged as an iron-deficiency anæmia. The effect of infections in reducing hæmoglobin is too well known to need much discussion. Pneumonia in infancy is almost invariably associated with some degree of anæmia, and sometimes the fall in hæmoglobin is considerable. Infections of the intestinal tract are rarely associated with great reduction in hæmoglobin. Especially important in infancy is the prolonged or repeated mild upper respiratory infection, which may not of itself cause any great degree of anæmia, but may yet prevent the hæmoglobin from rising to normal values. Such infections are probably an important cause of the low hæmoglobin in babies with poor hygienic surroundings.

Hygienic and Dietary Factors.

It is hard to estimate the effect of poor surroundings as a factor in the reduction of hæmoglobin. Bad hygienic surroundings include the probability of frequent exposure to infection, inadequate diet for the mother during pregnancy, and for the child later. Rickets is a disease of poverty in cities, and the association of rickets and anæmia is well recognised, even though the reason for the association is not yet understood. Much work has been done on the relation of the lack of vitamins to anæmia, but little is definitely known. There are cases on record in which the anæmia accompanying scurvy has been cured by the administration of orange juice alone, but clear evidence of the curative value of other vitamins is lacking.

There are other factors less commonly the cause of anæmia in infancy. Among these may be mentioned hæmorrhage—confined almost exclusively to the newborn period—lead poisoning, and injury from deep X-ray therapy. The symptoms of lead poisoning are often obscure, and anæmia may be an early indication of danger from this source. The anæmia following excessive use of X-rays is hypoplastic in nature, and is likely to be very resistant to treatment.

Clinical Characteristics.

The great majority of the cases of anæmia encountered in infancy are due to iron deficiency, the reasons for which have been given in the foregoing discussion. The symptoms are a mixture of those due to the anæmia itself and those associated with whatever it is that may be causing the anæmia. One of the most important of the symptoms is loss of appetite for anything but milk ; because of this the child deprives himself of the very food that might cure the anæmia. One of the first symptoms of beginning recovery, even before the hæmoglobin has begun to rise, is a return of appetite. Pallor is, of course, present, but is most misleading, and it is a mistake to attempt to diagnose the degree of anæmia without a determination of the hæmoglobin. It is extraordinary how well the infant tolerates severe anæmia: activity may be normal, and the parents may think nothing amiss even when the hæmoglobin is less than 30 per cent. Examination of the blood shows a hypochromic anæmia, with colour index usually 0.5 or below. While low colour index is usual, exceptions may occur, *e.g.*, the anæmia of scurvy, or during the course of a severe infection. Reticulocytes are moderately increased—4 to 5 per cent. at most—unless an infection is present, when they may be absent. Nucleated red cells are rare. Marked increase in reticulocytes and erythroblasts points to a more serious type of anæmia, hæmolytic in origin, or in which there is severe involvement of the hæmatopoietic tissue. Such types of anæmia will receive brief consideration later.

Treatment.

Prophylaxis of anæmia has been discussed under “ physiological anæmia ” and anæmia of prematurity. Enough has been said of the various factors to indicate the general lines of treatment. It cannot be too strongly stated that the first essential of treatment

is the diagnosis, and that the diagnosis of anæmia consists not in supplying a name, but in determining all the factors that may be responsible for the anæmia in order as far as possible to eliminate them and repair the damage they have wrought.

Treatment with Iron.

In the treatment of anæmia, iron has a two-fold function : (1) It stimulates the hæmatopoietic tissue ; (2) it supplies a " building-stone " for hæmoglobin. It should be given in large doses, 40–50 milligrams per kilogram of body weight per day. The form in which it is given is immaterial. I have found ferric ammonium citrate satisfactory made up in 10 per cent. solution and given in divided doses in the milk—2 c.c. per kilogram of body weight per day. After a latent period of four or five days the red cells and hæmoglobin begin to rise. The rapidity of this rise depends on the initial hæmoglobin, as well as on factors, such as persistence of infection, that might tend to retard recovery. Starting at 20 or 30 per cent., the rise in hæmoglobin may be extraordinarily rapid at first, but often tends to become slower after 50 per cent. is reached, so that from this point up to 80 per cent. recovery may require several weeks. In such cases, usually the result of infection, copper may speed the recovery. Copper appears to have a specific effect on hæmoglobin synthesis, but does not stimulate hæmatopoietic activity, and is indicated in cases in which hæmoglobin formation is slow, as shown by the persistence of a low colour index after the red cells have risen to normal levels. It may be given in a simple solution of copper sulphate. I have used 1/2 per cent. solution of the crystals in a dose of 1 c.c. per kilogram of body weight per day given in the milk. In infants this has rarely caused any digestive disturbance, but in older children or adults this amount of copper may cause nausea and diarrhœa. By starting with a smaller dose and gradually increasing the amount, a tolerance to the larger dose is easily established. However, copper is not essential to the treatment of anæmia ; it speeds recovery in some cases, and need not be continued for more than a week in any case.

Transfusion.

The rapidity with which the hæmoglobin rises after the administration of large doses of medicinal iron has rendered the treatment of the ordinary case of anæmia with transfusion unnecessary.

Even when the hæmoglobin is as low as 20 per cent. there is no need for immediate action, for anæmia at this age is ordinarily chronic, and the gain of a few days afforded by transfusion is hardly worth the expense and trouble entailed. The chief use of transfusion is to combat infection, or to put the child in a better condition to resist whatever factors may be responsible for a lack of vitality. It is the method of choice in hæmorrhagic disease of the newborn, but rather for the stopping of the hæmorrhage than for the raising of hæmoglobin; for once the hæmorrhage is controlled recovery is usually rapid.

Rare Forms of Anæmia in Infancy.

Anæmia of the Newborn.—This is not a disease, but rather a grouping of different types of anæmia occurring in the first few weeks of life, the causes of which are obscure or constitutional in origin. A few cases may be aplastic in nature, and for these there is no cure. Transfusion is palliative, but death is inevitable. The hæmolytic types with jaundice are usually temporary in nature, but owing to the rapidity with which the hæmoglobin may fall, transfusion must be resorted to immediately and repeated until the tendency to increased hæmolysis has ceased. The immediate prognosis is ordinarily good; the eventual prognosis is somewhat uncertain, but in the light of our present knowledge appears to be good. In a third group we know nothing of the factors, but the tendency to anæmia appears to be temporary, and is possibly only an exaggeration of the "physiological" anæmia of this period of life. Transfusion is indicated if the hæmoglobin is falling rapidly, but a number of the cases reported in the literature have recovered without treatment.

Constitutional Anæmia and Leukæmia.—A detailed discussion of these types does not come within the scope of this paper. In those cases in which there is increased hæmolysis without severe disturbance in the blood-forming process splenectomy offers hope of cure; but in others in which blood formation is profoundly disturbed, as indicated by the appearance in the blood stream of markedly immature myeloid elements, there is little hope of successful treatment. It is to this group that most of the cases of von Jaksch's anæmia belong. Certain of these have recently been separated out by Cooley under the designation of "erythroblastic anæmia" (called also "Mediterranean anæmia" because they

occur in children of the Mediterranean races). While such cases are clearly of constitutional origin, others showing the von Jaksch's picture may be the end result of a long-continued secondary anæmia in which the causative factor has eventually injured the hæmato-poietic function. In such cases the hope of permanent cure rests in the discovery and elimination of the causative factor. The elimination of the constitutional factor has thus far not been accomplished, though the anæmia may in some cases be greatly benefited by splenectomy.

Summary.

The problem of the treatment of anæmia in infancy is two-fold : (1) The discovery and elimination, as far as possible, of the factors responsible for the reduction in hæmoglobin ; (2) the treatment of the anæmia itself. I have attempted to show how the most common factors act to deplete the iron reserves of the body, so that in the great majority of cases the anæmia is eventually dependent on iron deficiency and is readily cured by large doses of iron. As long as the factors responsible for the anæmia are present it is useless to expect a cure of the anæmia ; but fortunately these factors are usually self-limited, or can be readily discovered and eliminated, so that at most all that is eventually left is the easily treated iron deficiency. In a small minority the anæmia is dependent on a severe disturbance in blood formation or destruction. The responsible factors in these cases are usually constitutional in nature. The treatment of these cases has been briefly discussed.

CHAPTER XL.

THE TREATMENT OF SOME OF THE COMMONER SKIN DISEASES CAUSED BY COCCI

BY

R. M. B. MACKENNA, M.D., M.R.C.P.

Hon. Dermatologist, Liverpool Stanley Hospital.

Two of the most important genera of the big family of bacteria known as Coccaceæ are the staphylococci and the streptococci. Parasitic bacteria from these genera cause many skin diseases, *e.g.*: impetigo contagiosa, furunculosis, erysipelas, coccogenic sycosis, Bockhart's impetigo, ecthyma, infantile pemphigoid, dermatitis gangrænosa infantum, hidrosadenitis, etc.

It may be of some interest to the reader if I attempt to describe briefly some of the methods which may be adopted in the treatment of the more common of these diseases. First :

Impetigo Contagiosa.

The standard casualty department treatment for this is ung. hydrarg. nit. dil., applied copiously. Impetigo is an acute infection of the skin, with streptococci and/or staphylococci, and is characterised by the effusion of a copious sero-purulent exudation from the affected area. If the usual mercurial ointment is applied on lint, it forms a waterproof covering under which the exudation may spread, thus infecting adjacent areas. Further, the fatty base of the ointment retards the normal heat loss from the skin, the temperature of the covered area rises slightly, the openings of the sweat and sebaceous glands are dilated, and a very fertile soil is prepared in which the cocci may increase and multiply in warmth and comfort. Cases of impetigo caused by relatively innocuous strains of bacteria may respond to the standard treatment with dilute

nitrate of mercury ointment, but if the infecting organisms are of a virulent nature, or the resistance of the patient is low, the condition of the patient rapidly goes from bad to worse. Good results may be obtained by the following line of treatment :

(1) Remove the crusts either by softening them with olive oil, or by the application, for six or eight hours, of a starch and boracic poultice.

(2) Apply the following lotion six or eight times daily :

R	
Sulph. precip.	gr. ii
Ichthyol	gr. iii
Calaminæ prep.	gr. lx
Zinci oxidi	gr. xxx
Glycerin.	m. x
Aq. calcis	℥iv
Aq.	ad ℥i
	M.

(3) Remove the crusts regularly once every thirty-six hours.

(4) When the exudation ceases and the only symptom is slight redness of the affected areas, apply the following ointment twice daily :

R	
Hydrarg. Ammon.	gr. v
Paraffin. Mollis	ad ℥i
	M.

It will be seen that the rationale of this line of treatment is to soothe the inflamed areas with a modified calamine lotion, to increase the formation of crusts, so that the exudation is localised, and to apply the lotion so frequently that the ichthyol and sulphur, and to a lesser degree the other ingredients, can exert their antiseptic action on every drop of exudate. Cases which do not respond to this line of treatment frequently respond quickly if the following lotion is applied freely four times daily to the affected parts :

R	
Hydrarg. Perchlor.	gr. 1/3
Sp. Vini meth. indust.	℥ss
Glycerini	m. v
Aq.	ad ℥i
	M.

Never forget that impetigo in children, commencing on the ears or in the scalp, is usually secondary to pediculosis capitis. If lice or nits are found, and the impetiginous eruption is at all widespread over the scalp, the best line of treatment is to cut the

hair short, and apply compresses of perchloride of mercury lotion (*vide supra*) thrice daily to the scalp.

Furunculosis is a disease caused by cocci, and frequently is difficult to eradicate. We should remember that in all cases of boils the urine must be tested before treatment is commenced. Even if there is no sugar in the urine the patient will benefit if his intake of carbohydrates is halved, and spicy foods, *e.g.*, hors d'œuvres, pickles, chutneys, high game, etc., should be taboo. There is one point with regard to furunculosis which is not usually emphasised, and that is that an attack of boils commonly develops after several months, or even years, of self-neglect, and unless the patient's life is regulated from all aspects, furunculosis frequently becomes distressingly chronic. The house surgeon, during the last weeks of his appointment; the student or the nurse working for his or her examinations; the schoolmistress towards the end of term; the adolescent struggling with higher school certificate, a mass of unresolved sex complexes and the frequent over-strain of team games; the typist or clerk who is habitually constipated and never takes exercise; the workman who suffers from chronic under-nourishment and lacks personal cleanliness—all these are types which often give a history that they started to have boils, and despite much treatment the boils have continued to erupt—often in crops—from time to time. If the physician is content merely with the usual local and tonic treatment, the condition of the patient frequently goes from bad to worse. Frequently the patient herself is her worst enemy. The stenographer will indignantly state that she is getting at least an hour and a-half exercise in the open air daily, looking after her bowels, and doing all things as she has been directed, and yet she is not better. She does not tell you that she smokes fifteen to twenty cigarettes a day, frequently takes aspirin in large doses, invariably reads in bed until one o'clock in the morning, and never sleeps with her window open. Similarly the workman will conscientiously go to the local bath-house thrice a week, and even have potassium permanganate baths; but if we have forgotten to warn him that it is no use attempting to disinfect the skin in this way, when on leaving his bath he dons an old and unwashed woollen vest on which is the coagulated excretion from the half-dozen boils from which he has previously suffered, then he will continue to suffer from furunculosis. These matters seem so simple that they are seldom referred to; frequently, however,

persons go on from month to month suffering from boils or carbuncles, and if the physician would only discuss fully his patient's habit of life, he would discover and be able to rectify apparently trivial matters which are frustrating the whole of his treatment.

The local treatment of furunculosis has been so thoroughly dealt with in various recent publications that I intend only to make brief reference to it here. The most modern line of treatment is by solutions containing antiviral, or jellies containing bacteriophage. For boils of the external auditory meatus, gauze plugs soaked in antiviral solution and changed three or four times daily are excellent, and I have had good results from packing the cavities of carbuncles with bacteriophage jelly; but for the average case of furunculosis it has been my experience that the benefit obtained by the use of polyvalent staphylococcal antiviral is in no way commensurate with the high cost of treatment. For the majority of cases I prefer to give non-specific therapy in the form of intramuscular injections (once weekly) of 1.5 c.c. butyrate of manganese or collosol manganese in the increasing doses recommended by the makers; concurrently, most cases benefit if given two tablets of oxide of tin (stannoxyl) thrice daily before food, and a drachm of Easton's syrup (B.P. 1914) thrice daily after food. Many authorities prefer to use vaccines rather than the preparations of manganese; an autogenous vaccine is frequently excellent; stock vaccines are successful in some cases.

One of the most dangerous diseases due to cocci is erysipelas; facial erysipelas frequently ends fatally. Recent work by Semon, McEachern, Sayed and others has indicated that injections of milk frequently benefit apparently hopeless cases. One such case, following as it did upon McEachern's careful report on this subject, particularly impressed me. The case was one of facial erysipelas following a graze on the bridge of the nose in a man of sixty years of age. Despite the subcutaneous injection of large quantities of both polyvalent antistreptococcal serum and anti-scarlatina serum, the patient's temperature had for a week swung between 101° to 103°; both cheeks, eyelids and the forehead were affected, and the spreading edge of the eruption was travelling rapidly over the scalp. At 1.30 p.m. on the seventh day of the illness, 10 c.c. of aolan (detoxicated milk protein—Beiersdorf) were injected intramuscularly. By 8 p.m. the patient's temperature had risen to 105°, but during the night the temperature dropped to normal,

and although no further injections were given, the temperature did not again rise, and the eruption rapidly disappeared.

Fresh milk may be prepared for injection by boiling for four minutes, cooling to body temperature, and straining through sterile gauze. Sayed has had good results by the intramuscular injection of 10 c.c. of milk in cases of facial erysipelas, and also claims that "intra-dermal injections of sterile milk made parallel to and about $1\frac{1}{2}$ in. away from the spreading margin of erysipelas produce a local reaction which prevents further extension of the infection at the site of injection, and improves the condition elsewhere."

Supplies of serum may be difficult to obtain in some country districts, but there is usually an adequate supply of fresh milk, and the practitioner faced with an acute case of erysipelas may find that even if he cannot manage an intra-dermal injection, an intramuscular injection of milk may influence very favourably a case which could otherwise give rise to much anxiety. If a case of erysipelas is going to respond to intramuscular injections of milk, usually only two or three injections are necessary, and many cases clear up after a single injection.

Cases of coccogenic sycosis are frequently extremely resistant. Many cases will respond to the following line of treatment :

(1) Clean up the face carefully, puncturing any pustules and expressing their contents. If a hair is visible at the centre of any pustule, remove it with a pair of forceps. Remove any crusts either by means of a starch and boracic poultice, or with olive oil.

(2) Instruct the patient to desist from shaving, but to keep the beard short by clipping or cutting with scissors.

(3) Fold some white gauze so as to make a pad about six layers in thickness, soak it well in the perchloride of mercury and spirit lotion (*vide supra*), and place this in close contact with the skin for half an hour at a time, and instruct the patient to carry out this procedure thrice daily.

(4) Remove the gauze compress after the half-hour has elapsed, and apply either calamine lotion with 1 per cent. ichthyol, or the following ointment :

R

Hydrarg. Ammon.	gr. iv
Ichthyol	gr. iv
P. Mollis	ad $\frac{3}{4}$ i

(5) If there is any blepharitis this must be treated on the usual lines, for the tear sac and the intranasal mucosa become infected with organisms washed off the eyelids by the tears, and the skin of the upper lip becomes constantly re-infected from these sources.

(6) General treatment includes avoidance of exposure to bright sunlight or cold winds, a wisely disciplined mode of life, tonics, and also a course of injections of butyrate of manganese.

If these measures fail, one may have recourse to such coal tar derivatives as acriflavine or brilliant green. Either may be used in an ointment (1 to 2 per cent.) or in a lotion (1 in 1,000). Some authorities recommend strongly alcoholic solutions of brilliant green. The following ointment, for the formula of which I am indebted to Dr. P. B. Mumford, occasionally cures cases of sycosis which have resisted other lines of treatment :

R	Sulph. precip.	℥i
	Balsami Peruviani	℥i
	Ung. Diachyli	℥i
	P. Mollis	ad ℥ii

M.

Sig.—Apply twice daily.

If these measures fail, then the practitioner will probably have to refer his patient to a dermatologist, who will decide whether or not the case is one which will benefit from epilation with X-rays, or from some other treatment.

Bockhart's impetigo is a superficial follicular suppuration which may arise spontaneously without apparent cause, or may follow the application to the skin of such irritants as tar, mercurial ointment, or turpentine. Any of the hair-bearing regions of the skin may be affected, but the thighs are the site of election. The best line of treatment is to open the pustules and apply a mild antiseptic lotion, e.g., a 1/5,000 solution of perchloride of mercury ; but before prescribing such treatment the physician must discover whether any of the above-named irritants have caused the disease ; if the eruption is due to the application of mercurial ointment, then a mercurial antiseptic is contra-indicated ; calamine lotion, with two grains of sulphur to every ounce, may be used instead, or if economy in dispensing costs is important, use :

R	Liq. aluminii acetici	℥i
	Glycerini	m. x
Aq.		ad ℥i

M.

Sig.—Apply to the affected parts on gauze thrice daily.

Ecthyma.—This may be considered as being a severe form of impetigo, the main lesions being pustular vesicles or bullæ. The bullæ or vesicles rupture, crusts form, and the lesions then appear as greenish-yellow or blackish-brown thick masses of dried pustular exudate, set on a hard, infiltrated and often painful base. The surrounding skin is erythematous. The sites of election are the buttocks, thighs and legs. The prognosis is good, but healing is usually slow, and scars of varying degree mark the sites of the lesions. The disease is streptococcal in origin, and may arise *de novo*, or as a complication of, *e.g.*, scabies. Children are more frequently affected than adults. Ecthyma usually arises in debilitated patients, and therefore an important part of the treatment is to improve the general health. A healthy environment and a reasonable diet are essential; a moderate amount of exercise may be taken once convalescence is established, but the majority of these cases should in the early stages be confined to bed. We should be careful not to miss an underlying and primary condition, *e.g.*, scabies, and if such condition is present it must be adequately treated. Whilst adult cases frequently respond favourably to injections of manganese, many children derive benefit from colloidal manganese taken by the mouth; the initial dose is 10–20 minims in water, thrice daily, and this may be increased slowly to 30 or 40 minims. Local treatment consists of removing the crusts and applying antiseptic lotions. American authorities strongly recommend local treatment with ultra-violet light, which they claim stimulates healing and stops the development of new lesions. Andrews recommends “first degree erythema exposures from a distance daily, or every other day, for several weeks, and this local treatment should be accompanied by generalised exposures for tonic effect.”

From the perusal of the above paragraphs, the reader may perhaps think that I have advocated standardisation of treatment for the various common coccal diseases, but I should like to emphasise that this is an error into which we must not be trapped; impetigo contagiosa in the slum child presents a problem different from that of impetigo in the well-nourished child of the middle classes; similarly, as I have indicated, furunculosis in various people affords different problems. If success is to be obtained the physician must bring all his knowledge of general medicine to bear upon the dermatological problem at hand: the patient's

general health, his reserves of strength, the foci of infection which he harbours, his habits and environment—these and all other matters must be considered before a course of treatment is prescribed. Treat him as a purely medical case, and co-incidentally treat his skin ; and always we should remember that weak strengths of non-irritant antiseptics applied frequently will do more good in contagious skin diseases than the occasional application of high concentrations of irritant antiseptics, which do more damage to the skin than to the invading bacteria.

CHAPTER XLI.

THE TREATMENT OF CONJUNCTIVITIS

BY

W. C. MACFETRIDGE, M.D.

Ophthalmic Surgeon, Sir Patrick Dun's Hospital and
Drumcondra Hospital, Dublin.

IN considering the treatment of conjunctivitis, it is first of all essential to bear in mind that the great majority of cases are due to bacterial infection. A few cases arise from other causes, such as smoke, irritant vapours, or errors of refraction, but the most common origin is microbic. As a rule it is not possible to say from a study of the clinical features of a case what the causal bacteria are, for a particular microbe does not produce the same clinical appearances in every patient; and if we want to identify the micro-organism we must call in the aid of a bacteriologist. However, from the point of view of treatment it is generally not of great importance to be able to say what the exact microbe is. But patients must be warned against the danger of infecting others. They must be told that the discharge from the eyes is infectious, and that towels, handkerchiefs and other such articles are a common way of disseminating the disease. The conjunctiva is a membrane, and it forms in the orbit a mucous sac. When it becomes inflamed pus and muco-pus are secreted and collect particularly in the folds and fornices of the conjunctiva. Our aim is to clear away all this purulent or muco-purulent discharge, and to destroy the invading microbes. It is a great mistake to put a bandage of any kind on a case of conjunctivitis. A bandage keeps the discharge in instead of helping to get it out; and by keeping the tissues bathed in pus it may easily produce serious damage to the cornea, which would be a very grave complication. It is not necessary to protect the eye from exposure to air. Fresh air is good for conjunctivitis, and patients should be encouraged

to go out of doors. Let us now consider some of the principal means of treatment. Lavage of the conjunctiva is useful, and is essential in every case where there is much discharge. It washes out the pus and microbes and offensive material of all kinds, and cleans the conjunctival sac. The best way to carry out lavage is with a pear-shaped glass vessel called an undine, but if this is not available, it can be done very well with a glass funnel or the barrel of a large ear-syringe with a piece of rubber tubing attached. Whatever apparatus is used, it must be something that can be boiled *in toto*. Any bland lotion or weak antiseptic can be used, such as boric lotion, hydrarg. perchlor., 1 in 5,000, or Condyl's fluid, 1 in 2,000, and the tepid lotion is directed in a gentle stream all over the eye and conjunctival sac, while the patient is instructed to rotate his eye in different directions so as to irrigate the various recesses as thoroughly as possible. This lavage is carried out two or three times a day, or, if there is much purulent discharge, as often as every hour.

Drops, or collyria, are an essential part of treatment. The most useful are zinc sulphate, argyrol, protargol and silver nitrate. They are made up in watery solutions, and are best prescribed in half-ounce quantities. One drop of the solution is dropped into the eye three times a day, or oftener, with a glass dropper that must never touch the eye and must be rinsed in boiling water before and after use. It is a good plan to put in the drops at meal time, as this regularises the treatment. A drop should never be put in at bedtime, for the resultant reaction may interfere with sleep, and increased secretion may be pent up inside the closed lids. If one kind of drop does not seem to suit a case we should not hesitate to try another. Some drops lose their efficacy after a time; the conjunctiva seems to have got accustomed to them; while other drops, such as argyrol, produce an irritation if used too long.

Zinc Sulphate is one of the most useful of all eyedrops. It has the great merit of being clean and cheap, and is a specific in one kind of conjunctivitis—the angular variety. But it is rather pungent, and nervous patients complain that it is painful, and will not use it for this reason. The usual strength is two grains to one ounce distilled water, but we may begin with a weaker solution, and if this is tolerated, work up to stronger solutions—as much as 1 per cent. A little boric acid is generally added.

Argyrol is a mild antiseptic collyrium, astringent, and not painful. Usually it is prescribed in from 10 to 30 per cent. solution, 25 per cent. being a good working strength. Like the other silver salts, it is supposed to have great penetrative power, but some authorities say that as a germicide it is negligible. Clinically it is beyond all doubt beneficial in many kinds of conjunctivitis, and as it is rather soothing, it often becomes a favourite drop with patients. But it is messy, gives to linen a temporary brown staining, and if used over a long period may produce argyria. No patient should be allowed to use it unless under frequent observation. A patient of mine for whom I had prescribed the usual 25 per cent. argyrol drops disappeared for three months, and used the drops diligently in the interval; he then returned with an intractable brown staining of his conjunctiva, and said his appearance was spoiled.

Protargol is usually prescribed 2 to 5 per cent. Its uses are very similar to argyrol. It is of a light-brown colour, and is less likely to produce argyria, but it may give pain. On the whole, it is not so generally applicable and useful as argyrol.

Silver Nitrate is the most active and powerful germicide we have. It is greatly used in 2 per cent. solution, to be applied to the conjunctiva of the everted lids in gonococcal cases, in trachoma, and in other severe forms of conjunctivitis. Sometimes it is prescribed as drops in 1/4 per cent. solution, but these should rarely be entrusted to patients, as there is always danger of argyria.

~ *Tannic Acid*, grain 5 to the ounce, is useful, and a mild astringent collyrium.

We will now consider briefly some of the most common kinds of conjunctivitis:

Gonorrhœal Ophthalmia.—In infants this is called ophthalmia neonatorum, and is a common cause of blindness. It is a most serious condition alike in infant and adult, and the gravest view of the case must be taken from the start. It is not uncommon, if the gravity of the disease is not appreciated, to see both eyes lost within a few weeks. The most expert knowledge and treatment are necessary. As soon as the case is seen, active and vigorous treatment must be put in hand. Lavage with weak Condy's fluid, 1 in 4,000, every hour, or at any rate every two hours, is indicated. All pus must be cleaned away, and the eyes kept as clean as possible. Silver nitrate in 2 per cent. solution must be applied every morning to the everted lids, and 1/4 per cent. drops instilled three times a day. The great

danger is infection of the cornea, followed by ulceration and possible destruction of the eye.

Trachoma.—This is a very serious and very chronic form of conjunctivitis. Even in the most experienced hands, and with every opportunity for treatment, the average duration of a case is from four to five years. In patients who do not attend regularly the disease is practically incurable. The main treatment consists in the application to the everted lids of 2 per cent. silver nitrate in the early stages, and of copper sulphate stick in the later stages. This is done three times a week. The many complications that may arise, *e.g.*, pannus, corneal ulcer, trichiasis, require special treatment. Patients should be warned as to the infectiousness of the disease.

Phlyctenular Conjunctivitis.—This is a disease of childhood and youth, and is the most common eye disease of children. It is thought to take its origin in the strumous diathesis, and undoubtedly is usually found in debilitated children. Treatment consists in application to the eye of unguent. hydrarg. ox. flav., 1 per cent., or of calomel powder once or twice a day; usually once is sufficient. The calomel is less irritating than the ointment, and may suit better in the early stages of the disease. If the cornea is involved, atropine must be added to the treatment. The general condition of the patient is very important. Fresh air and suitable food must be ordered. Cod-liver oil is very useful, varied perhaps with syrup ferri iod. The disease is very liable to relapses, and treatment should be continued for some time after apparent recovery.

Angular Conjunctivitis.—This type of conjunctivitis is frequently met both in the acute and the chronic form. It is particularly common in a mild chronic form, with a little white discharge at the angles of the eyes. The specific treatment is zinc sulphate drops, from 1/2 to 1 per cent., three times a day. If this is carried out, cure is practically certain. The silver salts are of little use in this condition.

Acute Catarrhal Conjunctivitis.—This, a muco-purulent form of conjunctivitis, is of varying severity, and is often due to the pneumococcus. Treatment consists in the employment of the silver preparations, such as argyrol, 25 per cent., three times a day during the acute stages, and when the inflammation is subsiding we may change to the astringent drops, preferably to zinc sulphate. If

in this form of conjunctivitis, or in any other form, the lids are stuck together at night by discharge, it is well to order a simple ointment to be smeared on the edges of the lids at bedtime. Boric ointment does very well for this purpose. The unguent. hydrarg. ox. flav. is too irritating. A very useful and agreeable ointment is hydrarg. perchlor., one part, to three thousand parts white vaseline.

CHAPTER XLII.

THE TREATMENT OF PNEUMONIA IN CHILDREN

BY

T. R. R. TODD, M.D., F.R.C.P. Ed.

Physician, Leith Hospital; Assistant Physician, Royal Infirmary, Edinburgh.

THE prime object in the treatment of pneumonia is to maintain the functions of the organism in as satisfactory a condition as possible, and let nature combat the disease; at the same time a close watch is kept for special phenomena which receive appropriate treatment as they occur.

The child usually lies quietly in bed breathing rapidly, and in infants the rate may be over sixty per minute. Some children are very restless and greatly distressed. Owing to the large amount of lung which is inactive, it is necessary to provide an abundant supply of pure air. A large room with, if possible, cross ventilation is desirable, but no draught should be permitted to fall on the patient. Direct sunlight need not be obstructed unless photophobia is present. The ideal temperature for the room is 60-65° F., and when this is maintained few bedclothes are necessary: probably a sheet, two blankets and coverlet being sufficient. When the child is quiet and the room temperature satisfactory, the only garment he will require will be a Shetland wool vest, which allows ventilation of the skin without a too rapid loss of heat. A restless child should have a flannel gown also. Hot-water bottles are more desirable than an increased number of blankets, which are heavy. The bottles, preferably rubber ones, should be separated from the patient by a layer of blanket, as well as the usual cover.

Where suitable conditions exist it is an advantage to place the patient out of doors for a few hours each day.¹ It is necessary to protect him from the wind, and to give some extra clothing in the way of a bed-jacket, blankets and hot-water bottles, but he

should not be burdened by too many clothes. Winter is not necessarily a contraindication to fresh-air treatment, but cold weather is. Other contraindications are fog, rain and a smoke-laden atmosphere. A maximum of seven hours exposure each day may be obtained from spring to autumn, but even a couple of hours will be found to produce a beneficial effect, which will manifest itself by a curtailment of the restlessness and a marked improvement in sleep; cyanosis is lessened and the pulse-rate drops. In some cases where anoxæmia is marked, as shown by restlessness and cyanosis, oxygen should be supplied in addition to the atmospheric air. The Davies and Gilchrist oxygen apparatus may be employed, but as it is expensive, and requires supervision, it will not often be available.² Quite good results can be obtained by leading the oxygen from the cylinder through a wash bottle containing hot water, and then by means of a soft rubber catheter to the nose. The oxygen should be turned on previous to placing the catheter in the nose, in case the onset of flow is so rapid as to disturb the patient. The rate of flow should be as rapid as is comfortable for the patient. The catheter is smeared with 10 per cent. cocaine ointment, and retained in position with adhesive strapping. Another method of leading in the oxygen is by means of the nasal tubes from the Davies-Gilchrist apparatus. Do not delay the use of oxygen until the patient is *in extremis*, but commence it on the first indication of cyanosis or respiratory distress, and thereafter use it continuously.

A warm sponging twice a day is necessary for the hygiene of the skin, but when the temperature rises above 103° F., a tepid sponge (90° F.) is advisable, which may bring about a reduction of from one to four degrees of temperature. Tepid sponging should be resorted to as often as necessary, but it is not usually required more than twice during the twenty-four hours.

Fluids are even of more importance than food, but in infants milk is food as well as drink. For a time breast-fed infants may be unable to suck: then the milk should be drawn off and fed with a spoon. Other infants will probably require the milk diluted more than usual, and extra fluids between feeds should not be withheld. For older children milk will also be the main article of diet, but will be supplemented by orange and lemon drinks, all of which are useful vehicles for glucose. This most easily assimilable of all foods should be given in most feeds. One or

two teaspoonfuls is a convenient dose. If there be any nausea or vomiting the glucose could be withheld for a few hours, and grey powder, gr. $\frac{1}{8}$, or bismuth salicylate, gr. ijss, given with each drink. Sometimes the bowels are sluggish, and then a nightly dose of grey powder, syrup of figs or liquorice may be used, but another method is to give small doses of cascara or Lixen (A. & H.), min. 5-15 t.i.d. An enema is sometimes more suitable. Diarrhœa is a troublesome symptom, and requires immediate attention in the form of a dose of castor oil or salts, followed by Kaylene or Kaolin in teaspoonful doses every two hours (this dose is suitable for a child of six). Bismuth salicylate, gr. v, with each feed, is another useful agent.

It has been shown in recent years that vitamins A and D have a definite anti-infective value. Although pneumonia is a rapid disease, and vitamins are in comparison slowly-acting medicaments, I believe we should use them, and give the patient the advantage of an increased resistance to infection, which increases his chance of recovery and perhaps accelerates his convalescence. Clinical experiment in another acute infective disease, *viz.*, measles, demonstrated that vitamins A and D concentrate given throughout the illness reduced the death-rate and incidence of complications as compared with the control cases.³

A toxic myocardium has always to be kept in view, and may be recognised by a rise in the pulse rate and a weakening of its force. When it does occur we should endeavour still further to husband the reserve of the heart. Alcohol in the form of whisky or brandy does cause an improvement in the pulse. Occasionally benefit has followed the intramuscular injection of camphor in oil, 1 c.c., or coramine, 1 c.c. Digitalis is of little or no value.

Cough mixtures are seldom required, and should not be used as a routine. Children under seven seldom bring up any sputum, but occasionally one is seen coughing repeatedly in evident distress, trying to loosen and expel some obstruction. In this circumstance an alkaline expectorant is of service: Soda bicarb., gr. v; pot. iodid., gr. ij; tinct. ipecacuanhæ, min. v; aqua chloroform ad dram i, p.r.n. When a sedative is required, try codeine, gr. $\frac{1}{12}$ to $\frac{1}{8}$ in jelly, pastille or mixture.

Restlessness and sleeplessness almost entirely disappear when the children are treated out of doors for a few hours, but when that therapy is not available one of the following may be tried:

Chloral hydrate, gr. iv, with soda bromid., gr. iv ; nepenthe, min. ij to v ; paraldehyde, min. x to xxv.

Meningeal symptoms sometimes develop when an upper lobe is consolidated but also occasionally when a lower lobe is involved. The symptoms and signs are the same as those of meningitis, but the cerebro-spinal fluid, although under slight pressure, is clear, and contains neither pus cells nor organisms. When pneumococcal meningitis does develop death follows in three days. The treatment of meningismus is symptomatic, and takes the form of cold compresses to the head, and sedatives. Lumbar puncture may be done to relieve pressure.

Convulsions occasionally usher in pneumonia in young children, and require immediate treatment. A hot bath is the most readily available, and should be used at once: mustard may be added, but always with discretion. While cold water is applied to the head, the temperature of the bath water must be kept up. If the convulsions be not relieved in about twenty minutes, chloral hydrate, five grains by mouth or ten grains by rectum, should be administered. The other two remedies next to be tried are lumbar puncture and chloroform anæsthesia. The pain from pleurisy requires to be relieved, and local application of heat seems to be beneficial either in the form of hot fomentations or linseed poultices, which, however, require changing about every three hours, or as Caloplast, which is left in position for twenty-four hours. Too tight a bandage retaining the poultice is not desirable, as one does not want to restrict the respiratory movements unnecessarily. Empyema is a common sequela, especially during the first three years. Before aspiration is attempted the needle, syringe and plunger should all be tested for accuracy of fitting so that satisfactory suction can be obtained. The skin and deeper tissues should be infiltrated with a local anæsthetic. As fine a needle as will allow the pus through should be used ; and as the pus gets thicker a larger bore needle will be required. Repeated aspirations are always necessary, and there are several methods in use. The simplest is to use a 10 c.c. syringe and needle ; an improvement on this is to have a three-way connection between the syringe and needle, which avoids the necessity of disconnecting every time the syringe is filled with pus. The Potain aspirator is liable to develop leaks at the joints, and the cannulæ are rather large. Professor Charles McNeil's method of closed or intermittent

drainage has advantages. The main features are the insertion of a special short cannula into the pleural cavity, through which, and accurately filling the lumen, is placed a self-retaining rubber tube through which the pus is aspirated, and in the intervals it is clamped.⁴ In most cases, however, open operation will eventually be required, but the period of aspiration is valuable, as it allows the pus to be shut off, while it also gives an opportunity for the general improvement of the patient after the pneumonia. The administration of large doses of vitamins A and D should be continued, and the diet can now be increased liberally.

Remarkably rapid recovery is noticeable in some children after the crisis, the physical signs clearing up in a day or two. There is also another group in which resolution is delayed beyond the average. In these cases the re-expansion of the lungs is assisted by the use of Bunsen's blow bottles; but if the child cannot be induced to take sufficiently deep breaths for this purpose, the respiratory centre can be stimulated by giving him CO_2 and O_2 to inhale through a funnel for about five minutes several times a day. Convalescence is assisted by ultra-violet light therapy, which can be commenced a day or two after the crisis. When empyema has occurred the dosage must be very carefully regulated each day.

Broncho-pneumonia.—Broncho-pneumonia in children is a much more serious condition, but the treatment is substantially the same as that of lobar pneumonia. The patient is usually a badly nurtured child, and has little reserve of strength to enable him to fight the disease, so that chances of recovery are poor from the outset. Respiration is always distressed, and the patient is frequently very restless. When conditions are favourable these patients should be treated in the open air for as long as possible each day. This is impossible in many cases, and then continuous inhalation of oxygen should be instituted from the time the patient comes under treatment; unfortunately this also is not always available. Are there any drugs then which can be used for this malady? S.U.P. 36 (B.D.H.) has now been used for several years in broncho-pneumonia and influenza, and in many cases I have seen, improvement has followed its use. The dose is 1 c.c. injected intramuscularly once or twice a day. Elbon (Ciba) is a respiratory tract antiseptic, and should be used in large doses, three to ten tablets per diem according to age. Creosote in doses of 2 to 5 minims, also hexamine with belladonna, are all well-used remedies.

When secretion is copious it is advisable to increase the dose of belladonna to the limit of tolerance. In broncho-pneumonia expectorant mixtures are frequently required, and they should usually contain ammonium carbonate for its stimulant properties. Some patients are troubled by a dry hacking cough, which is relieved quite rapidly by moistening the atmosphere with steam. In a large room or ward a half tent erected round the head of the bed is necessary, but in a smaller room it is not required. Patients who have been treated by steam, and those who have laryngitis, should not be treated in the fresh air. Almost every child with broncho-pneumonia requires alcohol, but the dose should not be large. For an infant under one year, 5-15 minims four-hourly should be sufficient, and during the next five years the maximum dose might be doubled. Strychnine is of distinct value in some forms of respiratory distress.

Acute Bronchitis.—During the early stages it is sometimes impossible to decide whether a child has bronchitis, pneumonia or broncho-pneumonia, so the treatment of the patient is as for the most serious condition. When the diagnosis has been settled in favour of bronchitis, the treatment is modified according to the severity of the illness. Expectorant drugs are used as in broncho-pneumonia, and steam is occasionally indicated. During convalescence, if the cough is persistent and without sputum, Brompton mixture, or a compound elixir of ephedrine, might be used with advantage. In all affections of the respiratory tract care should be taken whenever possible to avoid a smoky or foggy atmosphere.

REFERENCES.

- ¹ C. B. Ker: *Scottish Medical and Surgical Journal*, January, 1904.
- ² H. Whitridge Davies, and A. Rae Gilchrist: *Edinburgh Medical Journal*, May, 1925.
- ³ J. B. Ellison: *British Medical Journal*, October 15th, 1932.
- ⁴ C. McNeil: *The Practitioner*, July, 1930.

CHAPTER XLIII.

THE
TREATMENT OF TUBERCULOUS
DISEASE OF THE HIP

BY

H. F. MACAULEY, M.Ch., F.R.C.S.I.

Assistant Surgeon, Mater Misericordiæ Hospital, Dublin ; Surgeon to Cappagh
and Goole Orthopædic Hospitals.

THE classical symptoms of tuberculous disease of the hip-joint are well known, but it is not so widely known that these symptoms may be rather accurately mimicked by other lesions, and on occasions tuberculous disease presents an atypical symptom complex. Certain conditions, *e.g.*, Perthe's disease in its acute phase, can with certainty be differentiated by the radiographic pictures (though clinically a distinction is not infrequently impossible), while in others differentiation is only possible by biopsy or by the response to treatment. It therefore behoves the practitioner, when he has availed himself of the modern methods of diagnosis, radiographic examination, Wassermann test, and Von Pirquet or Mantoux reaction, and has his opinion thus substantiated, to treat the case in the first instance as one of tuberculous disease of the hip. An early and complete response to this form of treatment will segregate such confusing conditions as simple arthritis and synovitis. Occasionally, however, an early case of tuberculous disease, particularly if the focus is osseous and extra-articular, clears up to a degree under a few weeks of efficient treatment, and it is well therefore that the practitioner should keep the patient under periodic supervision for some months.

The first and most important aspect of treatment is to recognise that a case of articular tuberculosis is essentially a secondary manifestation of a concealed primary and active focus. The detection of the latter focus may be impossible, but its treatment

is none the less necessary, and is the "constitutional treatment" of tuberculosis. The main feature of this general treatment is accommodation of the patient as far as possible in the open-air both by day and night. If a sun balcony or shelter is not available nor attainable, a well-ventilated room with a southern aspect should be chosen. The body of the patient in his fixation apparatus should be exposed to direct sunlight, when it is available, and at other times an ultra-violet ray lamp may be utilised, and the irradiation, both by natural and artificial heliotherapy, should be general in preference to local. In addition to a generous dietary, the patient should be given cod-liver oil during the winter months, while during the summer it may be replaced by irradiated ergosterol, and if anæmia is marked, Parrish's syrup should also be given. In fact general treatment is on time-hallowed lines. Little new has been added from modern research, though at the present time a special dietary (Gerson's diet) is being investigated in Germany.

This general treatment is of no less importance than local treatment, and its efficiency can best be attested by the general appearance of the patient, condition of his muscles, a chart showing monthly weight variations, and by observation of the sedimentation rate of the red cells.

Local Treatment.

This will vary with the stage at which the patient first seeks treatment. The principles of treatment rest on the following general factors :

(1) Treatment must be sufficiently prolonged to allow all spasm, pain, tenderness and tendency to deformity to subside.

(2) During the acute and subacute stages the hip should be treated in the abducted position to prevent spontaneous dislocation.

(3) Immobilisation should be in a position of best functional use, in case the hip should become ankylosed. In the child the position about 15 abduction, foot straightforward, and no flexion ; in the adult less abduction and some 10 flexion.

(4) During the period of immobilisation a slight degree of flexion should be maintained at the knee-joint, knock-knee prevented, and the ligaments of the knee should be supported against uneven traction pull below the knee ; all these objects can be achieved by making a small plaster trough behind the knee. Prevent drop foot by a foot-piece.

(I) Early Case with Little Deformity.

While there are additional methods, only two can be recommended to the practitioner treating a case outside a special orthopaedic hospital :

(a) *Jones' Abduction Frame*.—This appliance must be made to measure for the particular patient. It is a desirable method if trustworthy supervision can be obtained at home, as it allows of easy inspection of the affected part, and local heliotherapy. It is important with this splint to keep the wings of the splint closely fitting to the body of the patient, and to have continuous and strong traction and countertraction maintained between the leg (adhesive plaster), extensions tied to the lower end of the splint, and the perineal straps around the groin. The traction is meant to be fixed, and is not to be released in the ordinary routine of nursing. Keep the splint raised off the bed by blocks of wood for the ventilation of the back and prevention of splint sores. The patient can readily be transported, as the splint is self-contained, *e.g.*, into a garden.

(b) *Long Plaster Spica*.—The plaster should extend from the costal margin to the toes on the affected side, and include the upper thigh on the unaffected side. Rigidity is given to the splint by joining the thigh pieces by a thick transverse rope of plaster. The leg, of course, is placed in the optimum position before the plaster is applied. Cut a wide opening in front of the abdomen, and keep careful watch of the circulation in the extremities for the first twenty-four hours. This method is very useful in a rapidly progressive and painful hip, and if properly applied is almost fool-proof as regards nursing. Its disadvantages are: inability to carry out general exposure to the sun, considerable muscle wasting, possibility of abscess development out of sight, and occasionally an individual idiosyncrasy to plaster.

With either of the above methods radiograms are advisable at three months' interval. When the radiogram shows the bone outline clear and distinct, not fuzzy as in the acute stage, calcification increasing, with favourable clinical symptoms, the hip is left free from fixation for a probationary period of four weeks. During this time the patient is free from restraint, but in bed, and should the patient use the limb freely and confidently, without tenderness or deformity supervening, the case is ready for ambulatory treatment.

(2) Disease with Deformity from Muscle Spasm.

The deformity is reduced by weight and pulley traction, counter-traction being made by tilting the lower end of the bed on blocks and using the patient's weight for this purpose. An average weight to put in the extension tin is the number in pounds corresponding to the age of the child in years, plus one. In a severe case, or when the patient is a small child, additional countertraction may be obtained by loops of flannelette bandage passed around either groin (where the flannelette is covered with oiled silk), and tied to the outside ends of the top portion of the cot. Traction is first made in the line of deformity, the leg being supported, if flexed, on an inclined plane or pillows, the angle of suspension being gradually lessened as deformity rapidly subsides. It is important to see when the leg is brought down straight on the bed that the correction is true, and not false correction with an associated lordosis. To be sure, apply Thomas' test, flex the sound leg fully on the abdomen, and if flexion has been corrected, the affected leg will remain flat on the bed. Two other methods have been advocated, but are not to be recommended in general practice: forcible correction under an anæsthetic, and Pugh's method of nearly vertical suspension. When the deformity has been corrected, the case reverts to the first category, and is treated on the abduction frame or plaster spica until the disease becomes sufficiently quiescent for treatment by ambulatory methods.

(3) Treatment of the Convalescent Case by Ambulatory Methods.

The commonly ordered Thomas' hip splint is not recommended for treatment at any stage of tuberculous hip, and its use should not be countenanced. Excluding it, the following methods are available: (a) Plaster spica, patten and crutches; (b) short plaster spica; (c) walking caliper. The choice depends on the site and extent of the lesion and adjudged soundness of the cure, and the home co-operation available. (a) Gives the best fixation, and should be used if recumbency for some reason or another has to be shortened, and the disease is in fact subacute; (b) is used where the bone is recalcifying, but there remains a tendency to adduction or flexion deformity. If the disease has been mainly acetabular, use method (b), and if of head, use method (c). During

the ambulatory stage the patient should be seen at intervals of about three months for two years at least.

Treatment of Complications.

Abscess.—Not every abscess clinically detectable needs treatment. A deeply-seated abscess with thick covering which appears indolent and does not rapidly expand should be watched. It will often disappear spontaneously. Large abscesses, increasing abscesses, or those with a thin covering should be aspirated before the overlying covering becomes red and thinned,—aspiration is too late then. Cleanse the skin with ether, paint it with picric acid in alcohol, and freeze it with ethyl-chloride at some distance in the healthy integument from the periphery of the abscess; pass a wide-bore needle attached to a 20 c.c. syringe with careful asepsis into the abscess, and aspirate the contents, or as much of the contents as can be easily withdrawn. Cover with a sterile pad, and apply a tight bandage. Re-aspirate when necessary. An abscess which has become secondarily infected should be incised and dressed like an ordinary acute abscess, but aseptic precautions must be thorough.

Dislocation.—An early dislocation, which, by the way, is always the result of failure to treat the case in an abducted position, can nearly always be reduced by steady and strong traction and counter-traction, either on an abduction frame or by weight and pulley. If the hip is active, painful, spastic, and has a relatively short history, then reduction of the dislocation can be confidently expected. An old unreplaced dislocation which has been present so long that the hip is fixed by adhesions, is generally painless and is irreducible. In such, treatment must be operative, and should be deferred until activity has long subsided.

Sinuses.—Their treatment is the treatment of the hip condition. Curettage is harmful; incision only advisable if drainage is not free through the sinus and there are symptoms due to retained secondarily infected pus, and the injection of Beck's plaster or Calot's paste does not seem of any benefit. Treat them antiseptically. The sinuses will heal when the underlying hip conditions heal.

"Grumbling Disease."—Here the activity never fully subsides, and every time restraint is left off deformity manifests itself, though mildly and painlessly. In such a case, where the patient

is in good general condition and the radiogram shows the bones relatively well calcified, the operation of "extra-articular" fusion is indicated. Here a bone graft is placed from the great trochanter to the ilium to act as a splint. Should adduction deformity be fixed, a subtrochanteric osteotomy may be done at the same time as the grafting operation. After operation a plaster exactly similar to the plaster advocated in the acute stage is applied, with the leg placed in that amount of abduction necessary to make up for any shortening present. After about six months in the post-operative plaster it is usually safe to let the patient up in a short spica.

CHAPTER XLIV.

THE SPA TREATMENT OF CHRONIC RHEUMATISM

BY

W. S. C. COPEMAN, M.A., M.B., M.R.C.P.

Hon. Physician, British Red Cross Society's Clinic for Rheumatism ;
Assistant Physician, West London Hospital (Children's Department),
and Hospital of St. John and Elizabeth ; Consultant, L.C.C.

THE spa treatment of chronic rheumatism is as old as the Roman occupation of Britain, as is evidenced by the remains at Bath and elsewhere, and is confirmed by certain passages in Cæsar's account of his campaigns. It must not be thought, however, that there has been no progress since these times, for the modern spa is in most cases not only continually studying the therapeutic needs and comfort of its patients, but is, in addition, now often a centre for research work. The chief British spas have since the War been entirely reorganised, and can stand comparison with those anywhere on the Continent of Europe for efficiency of diagnosis, treatment and extraneous attractions, both scenic and urban. Anyone who has not visited an English spa since this period would, I think, be surprised at the difference between conditions prevailing then and now.

There still exist medical and other persons who are firmly convinced that the spas are unscrupulous commercial undertakings organised on a quackery basis for no purpose other than their own ends. Such persons are, however, mediævally-minded.

It may be considered advisable, before speaking further of the actual spa treatment, to classify the forms of chronic rheumatism which are suitable for such treatment. The Ministry of Health classification of 1924 cannot, I think, be bettered for general use, and is as follows :

Group I.—Non-Articular Manifestations.—(1) Muscular rheumatism (myalgia), including fibrositis, pleurodynia and torticollis.

(2) Lumbago, classified alone on account of its occupational interest. (3) Sciatica and brachial neuritis.

Group II.—Chronic Joint Changes.—(1) Rheumatoid arthritis. (2) Osteo-arthritis. (3) Gout, acute and chronic. (4) Chronic joint changes, unclassifiable.

Every form of disease mentioned above will in most cases require physical treatment at some period in its course, and can therefore be considered as a suitable candidate for spa treatment if this is available.

I am often asked for a definition of spa treatment which will be accurately applied to any spa, irrespective of its site or particular indications. So far as this type of treatment applies to the treatment of rheumatism, it may perhaps be said that spa treatment is the application of physical methods in the ideal environment. Further than this one cannot go without being in a position to refer to some particular spa which may have been fitted by nature, or by man, with special advantages for this purpose. The various factors which will go to constitute spa treatment at its best will include the possibilities for rest and relaxation, with change of routine and all that this implies; suitable climate, soil, skilled medical attention, and physical and other treatment properly applied. These factors may be briefly considered separately.

Rest and relaxation are not always the natural accompaniments of a holiday away from home, as most householders know. Private domestic worries cannot always be eliminated, but the hotel-keepers and those who cater for the social side of spa life can do much to determine the health value of a spa visit from this point of view. Rest does not necessarily imply doing nothing: certain persons are constitutionally unable to do nothing. In the case of these people it may be conceded that rest may consist in their doing something different, but congenial, in pleasant circumstances and surroundings. For others, however, who require real rest, but are unable to obtain this at home, the spa environment and régime are invaluable.

With regard to climate, it may be said that the spas and health resorts of this country may roughly be divisible into two groups: the bracing or tonic climate (the majority); and the sedative or relaxing climate (mostly in the south-west). Those patients for whom the former indication would appear to be indicated may naturally be expected, all else being equal, to do better at a bracing

resort; whilst children, who seldom need artificial stimulation, and old people, or those of asthenic constitution, will be better for having the edge taken off their nerves by means of a more relaxing climate. Fuller information with regard to the varying climates of the English resorts will be found in the hand-book compiled for the British Health Resorts Association, at the beginning of the Medical Directory, or from Dr. Alfred Cox, at its headquarters at 199, Piccadilly.

Soil seems to be of secondary importance—provided that the surface drainage of the district is adequate. Where the ground is level the subsoil should be of a permeable nature, such as sand or gravel, to allow of moisture sinking well down, and so allowing of a dry climate. Where the resort is on a slope the subsoil may be of clay or chalk, for surface water will be directed to the lower slopes, whilst winds will be more usual, and will also help to dry the atmosphere.

The physical treatment indigenous to a spa will depend principally on the natural waters of the place. These may be used either internally or externally, according to their composition. In most spas nowadays all the usual auxiliary methods of physical treatment, such as massage and electrical treatment, are also available.

The internal administration of the natural mineral waters of a spa should always be left in the hands of a local practitioner who understands their effects and peculiarities. There seems no doubt that these waters must be taken at their source if real benefit is to be reaped from them. It is at those spas where the waters are ingested that it seems reasonable to suppose that their exact composition is of the greatest importance. The chief chemical constituents of British spa waters are radium, sulphates and other "salines," iron, sulphur and iodine. Many of these waters are also used for colonic irrigation.

The external application of mineral waters by various methods is a most important aspect of spa practice, and is embodied in the science of medical hydrology. The exact composition of the waters used externally seems to be of lesser importance than when they are taken internally, if we may judge of the success of these methods when employed in town clinics using the local water supply. The methods employed comprise immersion in baths, with or without the addition of underwater douches, the "Vichy" or "Aix" methods

of muscle massage under running hot water, and progressive training of stiffened joints for increased function and relaxation of muscular spasm in "deep pool" baths of mineral water. Heat, massage and movement are the basal factors in all forms of physical treatment, both wet and dry; and water, being a very convenient medium for applying any or all of these factors, may be said to constitute the highest common factor of this important, although still somewhat neglected, branch of medicine.

The auxiliary methods of treatment do not vary in spa practice from the form in which they are employed elsewhere. Dry massage, as apart from the forms employed under water, as mentioned above, has its indications, whilst most electrical methods, such as radiant heat, infra-red ray and diathermy, aim at the application of dry heat where this is considered preferable to moist heat. Ultra-violet light is a useful tonic, and may be used where supplies of the natural article fall short of requirements. The standard of treatment in British spas has risen immensely since the War, and is now unrivalled anywhere abroad. Some of the more progressive spas, notably Harrogate, are now employing the highly-trained masseurs of the C.S.S.M.G. for the "wet" as well as the "dry" treatments.

The indication for spa treatment in rheumatic cases includes all the constituents of the classification given above at certain periods in the disease. Pain is not primarily an indication for such treatment, since this can equally be dealt with in towns. Completely crippled cases are unlikely also to benefit notably from a short sojourn at a spa. Cases where the restoration of function appears possible, however, and where the home environment is for any reason unsuitable, or where the patient has been under treatment successfully at home and appears to be getting somewhat "stale," will be amongst the best indications. Senility is a contraindication.

The medical personnel of a spa is all-important, and from this point of view the British spas are mostly very well provided. It must not be thought, however, that all medical men who are practising at a spa are necessarily specialists in spa methods, or are even interested in the matter. It is worth while before referring patients to these places to find out the names of the leading men who do specialise in these forms of treatment, if the best results are to be obtained. Such men will, in addition, co-operate loyally

with the referring practitioner, and return patients at the end of their course with progress notes and suggestions for "follow-up" treatment. This is not always my experience where patients are referred to certain foreign spas.

The "season" for the spas is roughly between May and the middle of September, although Bath, by virtue of its particular climate, is open during the winter in addition. The average length of a course of spa treatment is three to four weeks. If possible, a further week's holiday should always be prescribed subsequently, in order that the best results may develop and the fatigue of treatment be recovered from.

The British spas which are most suitable for the treatment of rheumatism are the following (in alphabetical order): Bath, Buxton, Droitwich, Harrogate, Leamington, Llandrindod Wells, Strathpeffer, Trefriw Wells and Woodhall Spa. The three latter are small and quieter places than the former, which are mostly towns of some size. Each has its own particular attractions, and the climates vary considerably, as does the composition of the waters.

The cost of treatment in England is often commented upon unfavourably in comparison with foreign spas. This has, however, been considerably reduced since the War, it being possible in several of these spas now to receive three weeks of full treatment for three guineas. In addition, the high standard of the actual technique now insisted upon in England, and the cost of travelling to a foreign spa, must all be considered.

Many practitioners are anxious that their poorer patients shall have the advantage of spa treatment, which was at one time considered to be the prerogative of the rich only. Various schemes are now extant whereby this may be achieved. The H.S.A. has an arrangement with certain of the spas whereby their members may be treated at a very low cost. Many of the leading men at these places are upon their "panel," and have agreed to give their services at a very reduced rate. In several spas, notably Bath, Buxton and Harrogate, mineral water hospitals of considerable size and up-to-date outlook have long been in existence, and admission may be procured into the wards of these institutions for suitable cases on application to the secretary of the hospital selected.

The London County Council has also an arrangement with Droitwich whereby selected cases may be referred there for brine-bath treatment at a very low rate of maintenance charge.

From what has been said it will, I hope, be realised that the spas are "moving with the times," and that there is a real effort being made to "deliver the goods" medically to all classes of patients, *via* their medical attendants. There is considerable evidence that the profession as a whole is slowly losing its blind faith in exclusively medicinal methods of treatment, and is turning more and more to physical methods for help in their never-ending war with chronic rheumatic disease. This being so, it seems inevitable that increasing demands will be made on the medical resources of the spas in the near future, as the private practitioner learns to use and value them.

The full particulars of any spa can always be obtained by application to the spa director at the various pump rooms. Medical men who visit a spa, and who wish to learn something of its working, are also, in my experience, always made welcome, and generally go away wiser than they come.

CHAPTER XLV.

SOME COMMON EYE DISEASES IN ADULTS

BY

A. H. LEVY, F.R.C.S.

Surgeon to Central London Ophthalmic Hospital.

THE eyeball, being seated relatively deeply in the bony orbit and protected by the lids and orbital margin, is less subject to external influences than the more superficial structures. Hence the affections which are most frequently seen are injuries and diseases of the lids, conjunctiva and superficial structures of the globe. For our purpose here it will be better to deal with these tissues, and just mention the few more deeply-seated conditions which are important from the differential diagnosis point of view.

Marginal blepharitis will be discussed in the next chapter on "Eye Diseases in Children," and all that is stated there applies equally to adults. These inflammations are frequently associated with an eczematous condition of the neighbouring skin, which must be treated at the same time.

Hordeolum is an infection of the follicle of a cilium, and the removal of the affected eyelash is generally sufficient to cure it.

Chalazion is an affection generally of the Meibomian glands, probably due to retention of secretion complicated by an infective agent, most frequently the *Staphylococcus aureus*. Chalazia are best removed from the conjunctival surface by an incision at right angles to the lid margin. An incision parallel to the lid margin should not be made, as this will cut across neighbouring glands, and thus lead to further retention cysts. After the incision the contents should be thoroughly scraped out, and as this is a painful process, it should be preceded by an injection of 2 per cent. novocain, or preferably novutox, into the tissues surrounding the swelling.

Ingrowing Eyelashes.—Apart from the gross cases, the result of trachoma or of the more severe forms of blepharitis, eyelashes may occasionally deviate in direction from the normal and, bending backwards, impinge upon the eyeball and give rise to irritation, chronic conjunctivitis, or even keratitis. Occasionally a hair may grow from the caruncle and give rise to similar irritation. It usually suffices to remove the offending eyelash with a forceps, the succeeding lash generally growing in the normal direction. If not, the follicle had better be destroyed by electrolysis. The negative needle is inserted into the follicle, and a current of 3 to 5 m.a. passed for about one to two minutes.

Conjunctivitis.

The affections of the conjunctiva are the commonest forms of eye trouble, and can be exceedingly troublesome. They may be divided into two main categories: physico-chemical reactions and infections. Foreign bodies may irritate the conjunctiva by their mere presence, or they may also contain chemical irritants, as, for instance, aniline dyes. A bit of the lead of an indelible pencil can cause extensive sloughing if allowed to remain in for any length of time. Certain organic substances act in the same way, as, for instance, certain pollens in the production of hay fever.

The infections of the conjunctiva are many and various, but for practical purposes can be divided into the acute and chronic forms. The commonest form of acute conjunctivitis is that due to the pneumococcus, and will be described in the next chapter. A less common form is that caused by the Koch-Weeks bacillus (a very small Gram-negative bacillus). This may occur in epidemic form, but, as such, is rare in this country. Treatment should be continued for a long time, and consists of cold lotion, such as oxycyanide of mercury, 1 in 8,000, with occasional applications of 2 per cent. silver nitrate. Argyrol drops, 10 per cent., are also very useful. To prevent the lids sticking together any indifferent ointment smeared on the edges of the lids at night suffices; but if the condition becomes chronic, then the insertion into the conjunctival sac of 1 per cent. or 2 per cent. yellow oxide of mercury is very helpful.

Chronic conjunctivitis can be subdivided into many forms, but the most frequent is caused by the Gram-negative diplobacillus of Morax-Axenfeld. Characteristic of this type is the redness of

the lids, frequently both conjunctival and skin surfaces, and limited more or less to the canthi. Patients complain of itchiness, the feeling of sand in the eye, and stickiness of the lids in the morning. Zinc sulphate is specific for this form of conjunctivitis, used as an eye lotion of the strength of one grain to the ounce, or as drops of double that strength. It is to be remembered that this form of conjunctivitis is very prone to recur, and therefore the treatment must be prolonged for at least six to eight weeks.

Lacrymal Apparatus.

An increased flow of tears can only be caused by two conditions: hypersecretion of the gland or interference with the outflow passages. Hypersecretion of the gland practically always means some irritation of the eyeball—from a foreign body on the cornea or under the upper lid, to an inflammation of the cornea or internal structures of the globe. The cause must be found and treated, whereupon the watering of the eye will cease. Interference with the outflow of tears may be caused by the impossibility of tears entering the passage, or by some mechanical hindrance in the canaliculi, the tear sac or the naso-lacrymal duct.

Under the first heading there is primarily malposition, especially of the lower lid: an eversion which carries the punctum away from the globe, and which if allowed to persist will lead to a definite ectropion. This condition is seen in flaccid lids, especially in elderly people, facial paralysis, cicatricial contraction, etc. An occasional cause of obstruction to the outflow of tears is the impaction of a cilium in the lower punctum.

If there be no interference with tears entering the passage, the obstruction must be more deeply seated, and may be of many different kinds and in various situations. The localisation of such obstruction can be gauged by pressure over the tear sac. If there be regurgitation through the canaliculi into the conjunctival sac, then the obstruction must be below the point of pressure. If there be no regurgitation, then the site of the obstruction must be proximal to the point of pressure, *i.e.*, along the canaliculus or at the point of entrance of the canaliculus into the sac. There may be a stricture from an old ulceration in the duct, or as the result of some periostitis. Passing a medium-sized lacrymal probe after a preliminary dilatation of the punctum will show the point of obstruction, and by dilating any stricture cure or relieve

the condition. If the sac has been the seat of prolonged inflammation, then pockets full of pus and diverticula will have formed, and more serious surgical procedures must be undertaken.

Cornea.

Foreign Bodies.—In searching for foreign bodies it is essential to have good illumination, with the light falling obliquely on the cornea, and a loupe through which to examine the cornea. It is well to instil cocaine first so as to eliminate the watering and the photophobia. In searching for a foreign body the upper lid should always be everted, as frequently foreign bodies get lodged in this position.

Superficial and Deep Keratitis.—There are many forms, some of which will be described in the next chapter. In adults the commoner forms of keratitis are those associated with injuries and infections of the cornea directly, or extension of inflammatory processes from the surrounding conjunctiva, as in trachomatous ulcers and pannus. And there is also that form which is associated with nerve lesions, as in peripheral neuritis, herpes febrilis, herpes zoster ophthalmicus and surgical interference with the Gasserian ganglion.

A common form which merits special notice is the hypopyon ulcer. This is a superficial ulceration of the cornea in which the anterior chamber becomes partially filled with sterile pus, and which clears up when the ulcer heals. The best treatment is to scrape the base of the ulcer with a small sharp spoon, and then with a finely-pointed match-stick to apply pure carbolic acid to this surface, being careful to allow the acid to penetrate underneath the overhanging edge of the ulcer, frequent douching with a mildly antiseptic lotion, and using an occluding bandage.

Herpes Zoster Ophthalmicus is always to be regarded as a serious affection. As long as the eruption is limited to the distribution of the supra-orbital nerve the treatment will be the usual one, but in addition it will be well to keep a close watch on the eyeball, and to instil atropine once or twice daily to minimise the effect of any cyclitis which may appear, and which will be mentioned later. If, however, the cornea should become the seat of an herpetic ulcer, the condition immediately becomes very serious. At the best the healing process will leave a scar which will almost certainly impair vision, and at the worst the ulcer will lead to perforation

of the cornea and possibly destruction of the eyeball. It is to be noted that the cornea is only involved in those cases in which the area supplied by the nasal branch of the ophthalmic division of the fifth nerve shows the herpetic vesicles. The reason for this is that the ciliary ganglion from which the eye derives its sensory fibres is itself supplied by twigs from the nasal nerve as it passes across the orbit. The nasal nerve supplies the mucous membrane of the nose and the area of skin comprising the tip and alæ of the nose. Hence when these parts are involved the eye must receive special attention. The treatment must be directed to protecting the cornea as much as possible: an occlusive bandage, warm bathing and atropine, 1 per cent., with possibly rest in bed.

Beyond the direct involvement of the eye in the herpetic process, a deep-seated cyclitis is frequently seen in all forms of herpes of the ophthalmic division of the fifth nerve. This is characterised by tenderness over the ciliary region, redness, and usually many precipitates on the posterior surface of the cornea (K.P.). As a rule this is a very resistant form of cyclitis, and requires prolonged treatment.

In all herpetic affections of the eyeball it must be remembered that the sensibility of the cornea is considerably reduced, so that the normal protective mechanism functions badly. Foreign bodies may remain on the cornea and give rise to ulcers, and there occurs superficial keratitis of a more or less intractable or recurrent nature. In addition there are active destructive processes analogous to those seen on the cornea after removal of the Gasserian ganglion, or its injection with alcohol for trigeminal neuralgia. All these types of disorder are best treated by complete occlusion of the eyeball, even to the extent of sewing the lids together. To do this it is necessary to raw the edges of the lids, and to place the sutures so that they do not come into contact with the cornea.

Sclerotic.

Scleral tissue is analogous to the tissue of tendons, and the inflammations affecting it are similar; characteristically in scleritis there is local thickening and hyperæmia. This thickening may go on to the formation of a definite swelling, which practically never softens or breaks down, and may last for weeks or months, and finally disappear. Recurrences are frequent, but as long as the changes are limited to the superficial layers (episcleritis)

no great harm is done. It is only when the major part or whole thickness is involved that there is left a localised thinning of the sclerotic which may lead to local ectasias. The treatment consists of hot fomentations, and the application of one or two per cent. yellow oxide of mercury ointment. The general health should receive attention, and any septic focus treated.

Iritis and Irido-Cyclitis.

The whole uveal tract—iris, ciliary body and choroid—is a vascular structure partaking of the nature of a serous synovial membrane, and is subject to inflammations of the same type. It is rare to get an inflammation of one part only, although certain types tend to involve one or other part more intensely than the rest. It is difficult in any given case to be certain of the exact cause, but the principal things to remember are chronic gonorrhœa, septic foci in the teeth, tonsils, etc., and, after that, tuberculosis and syphilis. The symptoms of the commoner forms of iritis are ciliary injection, pain, and the formation of synechiæ. The pain is due to the swelling and constant movement of the iris. The ciliary injection is a reddish-blue area just outside the corneal limbus, and is due to engorgement of the ciliary body. The synechiæ are the result of the plastic exudate from the inflamed iris which causes it to adhere to the anterior capsule of the lens. In the treatment atropine is the sheet anchor. It stops all iris movements, and thus quickly relieves the pain, and it dilates the pupil so that its margin is no longer in contact with the lens capsule, and thus obviates the formation of synechiæ. Hot fomentations are a powerful adjuvant, and beyond this the treatment of the underlying cause must be undertaken.

Glaucoma.

Any eye in which the intraocular tension is raised above the normal is termed glaucomatous. There are the primary cases in which the ætiology is obscure, and the secondary forms in which it is due to alteration in the nature of or some interference with the normal outflow of the aqueous fluid. If the rise of tension be acute then there occurs pain, which may be exceedingly severe, congestion and dilatation of the pupil. If the rise be slow, then there will be no pain, but only a slowly progressive loss of field and, later, diminution of visual acuity. The treatment in the acute forms is operation; in the chronic forms miotics, such as eserine, or operation.

The more deeply seated inflammations of the ciliary body (cyclitis) and choroid are, generally speaking, due to the same causes as iritis, and the treatment should be along the same lines. In this connection it is necessary to mention K.P. This is a deposit of fine particles on the posterior surface of the cornea, and is symptomatic of cyclitis. The inflammatory exudate from the ciliary body is carried by the aqueous into the anterior chamber, and in the course of the ordinary circulation of the aqueous fluid comes into contact with the cornea and, being sticky, adheres to it. In all inflammations of the interior of the eye careful search should be made for these deposits, as their presence helps greatly in determining the seat of the inflammatory change.

Iritis is occasionally confused with glaucoma, acute or chronic. The differentiation of these two conditions is of fundamental importance, since the treatment of glaucoma with mydriatics will inevitably damage the eye, and the treatment of iritis with miotics will increase the pain and lead to the formation of dense synechiæ. The differentiation is all the more difficult inasmuch as the tension of the eye may be raised in iritis by the increase of the quantity of fluid in the eye, and its albuminous nature. The main points are that in iritis the pupil tends to be contracted, while in glaucoma it tends to be dilated. The field of vision will be contracted in glaucoma particularly on the nasal side. The cornea in glaucoma tends to be hazy and the aqueous clear, while in iritis the cornea may be clear, but the aqueous will be muddy. In cases of doubt the instillation first of cocaine and later of homatropine will help to clear up the diagnosis.

CHAPTER XLVI.

SOME COMMON EYE DISEASES IN CHILDREN

BY

A. H. LEVY, F.R.C.S.

Surgeon to Central London Ophthalmic Hospital.

IN infancy, the earliest pathological conditions seen are those associated with the birth and the accompanying injuries or infections. The injuries are of varying severity, ranging from contusion of the lids to avulsion of the whole globe. Besides blood effusion into the lids, there occurs not infrequently subconjunctival hæmorrhages, which may be limited to the limbus, forming a red ring round the cornea, or may be so extensive as to involve the whole subconjunctival space. These conditions are of no great importance, and disappear spontaneously very quickly. A frequent injury is damage to one or other sixth nerve, leading to paralysis of the corresponding external rectus muscle. This may be mistaken for a convergent strabismus, but is to be differentiated from it by the fact that the affected eye cannot be moved outwards beyond the middle line and the squint position is only present when the child looks towards the affected side.

Infections.

The infections are more important, and may be caused by many different organisms. From its severity and the possibility of serious after-effects, the gonorrhœal infection has loomed largest, but it is not the only one, and some of the others can be very troublesome. There is no doubt that the incidence of gonorrhœal ophthalmia in the newborn has materially diminished in recent years; and since it has become a notifiable disease, and the treatment more or less standardised, the results have been very favourable. It used to be said that the prognosis in this disease was

favourable in inverse ratio to the wealth of the parents. In other words, that excessive treatment was harmful.

The guiding principle must be removal of discharges and protection of the cornea against injury : simple lotions to wash out the eyes, and the occasional instillation of a drop of 20 per cent. protargol. But in no circumstances must any solid substance be brought into contact with the cornea, whether it be fingers, swabs or instruments. The use of the Cr  d   method is still valuable, but perhaps the use of mercurochrome or 20 per cent. protargol would be just as effective and less irritating than the usual 2 per cent. silver nitrate.

Of the other infections, that with a long-chain streptococcus is the commonest, and can be very chronic. The most useful lotion in these cases is 1 in 8,000 oxycyanide of mercury, and the daily instillation of a drop of mercurochrome. Many of these cases of chronic conjunctivitis are complicated by either occlusion of the puncta lachrymalia or definite infection of the tear sac. The occlusions are easily overcome by the insertion of the point of a lacrymal dilator. Infections of the sac will necessitate probing and syringing under a general an  sthetic, but as a rule clear up very quickly. In older children the commonest form of acute conjunctivitis is that known as "pink eye." This is an infection with the pneumococcus lanceolatus. It occurs sporadically among adults, but most frequently as epidemics in schools and elsewhere. It is characterised by swelling of the conjunctiva with much discharge. It runs an acute course, reaching its height in three to five days, and then terminates by crisis quite suddenly, and in twenty-four to thirty-six hours the whole process comes to an end. If seen early the infection can be aborted by painting the whole of the inner surfaces of both lids with 2 per cent. silver nitrate. Thereafter, and in all cases seen late, cold boric acid lotion should be used frequently, and precautions taken to prevent the spread of the infection. Complications are infiltration of the superficial layers of the cornea, with possibly the formation of ulcers. These generally heal after the acute stage has passed.

Phlyctenular Conjunctivitis.

A very troublesome complaint in children is phlyctenular conjunctivitis, the characteristic feature of which is a typical efflorescence, the so-called phlycten. A localised redness develops

in the conjunctiva close to the limbus corneæ. In the centre of this reddened area a small greyish-white nodule appears lying beneath the epithelium, which can be seen to be raised over it. This nodule attains its maximum size in a few days, the contents become more milky, and are then emptied through the softened epithelium. A small pit or ulcer remains for a few days, but this soon fills up and becomes covered with epithelium, and the redness gradually disappears.

This is the typical course of an isolated phlycten, but recurrences are common, and in many cases multiple phlyctenulæ develop, so that the whole eyeball becomes reddened. While each individual phlycten is a short-lived phenomenon, the disease may last for months or years, especially if the cornea becomes involved. This may occur by direct extension of a limbal inflammation, or the process may start in the peripheral parts of the cornea, and give rise to marginal ulceration. Occasionally an infiltration will commence at or near the limbus and travel towards the centre of the cornea, carrying with it a leash of blood vessels, the so-called "wandering phlyctenule" or fascicular keratitis. In other cases superficial infiltrations form in various parts of the cornea, rapidly giving rise to small ulcers, and gradually becoming vascularised from the vessels at the limbus. This tendency to vascularisation is typical of the whole group of so-called scrofulous diseases of the eye. In the more severe cases the lid margins may be involved, and the skin show eczematous or impetiginous areas with the presence of rhagades. The causation of this disease has been a source of considerable controversy. It occurs in children of a tuberculous or scrofulous diathesis, but the efflorescence itself is not tuberculous.

The treatment must be both local and general. The most meticulous cleanliness must be observed, and all sources of irritation removed. The eye must be kept clean by means of simple lotion, such as glycerine thymol co. Astringents and caustics are to be avoided, or only used very sparingly when the lid margins are involved. The yellow oxide of mercury ointment should be applied regularly both to the lid margins and in the conjunctival sac. It may be remarked here that for ophthalmic use the ordinary B.P. preparations are of no use, or rather are harmful. Specially prepared ointments for eye use should be employed, such as those made by Messrs. Allen & Hanburys. In the event of the cornea

becoming involved, atropine is indicated. This is better administered as an ointment, either by itself, or incorporated into the yellow oxide of mercury. The general treatment must be directed to building up the strength of the child : a nutritious diet, but with very little or no meat, must be given, and tonics such as the glycerophosphates or the iodide of iron should be administered ; recently considerable success has been obtained by the use of vitamin preparations, such as ergosterol. Patience and perseverance are required, and it is well to continue the use of the mercury ointment along the lid margins for months after all the symptoms have subsided.

Inflammatory Lesions of the Lids.

An analogous condition of the lids of children and adults is blepharitis marginalis or ciliaris. This is a generic name for various inflammatory lesions of the lid margin. Hyperæmia and slight œdema of the edges of the lids occurring after prolonged eye work, or on exposure to cold winds, may be considered as the mildest form. A characteristic type is that arising as an extension of a diplobacillary conjunctivitis known as blepharitis angularis, because the angles of the lids are reddened and raw looking. In the so-called blepharitis squamosa there are dry, with occasionally fatty, scales between the ciliæ. When these scales are removed the lid margin underneath is found to be reddened, but not ulcerated, as in the case of blepharitis ulcerosa. In this latter condition the lid margins at varying intervals are occupied by circular ulcers or small abscesses from the centre of which one or more thickened cilia project. Loss of ciliæ occurs in both the squamous and ulcerous forms, and bald patches may be left. The more severe forms are to be regarded as serious diseases which require careful and prolonged treatment. This treatment consists in local and general measures. The first thing is to correct carefully any error of refraction, even of the smallest amount, and to make certain that the tear passages are clear. Zinc sulphate drops, one to two grains to the ounce, clear up any diplobacillary conjunctivitis and the consequent blepharitis in a relatively short time. The other forms require regular and careful cleansing and the application of a mercurial ointment, such as white precipitate or yellow oxide. The ulcerous form calls for thorough epilation and the application of 2 per cent. silver nitrate, or even touching of the ulcers with

the mitigated nitrate stick ; but patience and perseverance are required to bring about a cure. Persons subject to these conditions should be instructed to pay special attention to their lids all their lives, more particularly the regular cleansing of the lid margins by means of soap and water, and then leaving them perfectly dry. General treatment must be employed in cases requiring it, and special attention paid to the condition of the teeth and tonsils.

Diseases of the Cornea.

Affections of the cornea have already been mentioned incidentally to conjunctival disease, but there are some primary diseases of this structure which are particularly frequent in children, the most important being interstitial or parenchymatous keratitis. The characteristic feature of this condition is, in the first place, the appearance of infiltrated areas in the substantia propria ; and, secondly, the invasion of the deeper corneal layers by blood vessels. The infiltrations are due to the destruction of some of the fixed cells of the cornea followed by an influx of leucocytes. These small masses become confluent, and together with the destruction of more lamellæ, form widespread areas of opacity which are situated at varying levels in the cornea. The necrotic detritus is absorbed, the remaining corneal cells begin to multiply and to grow into the spaces left by the absorbed material, and then comes the ingrowth of new blood vessels, accompanied by newly-formed fibrous tissue. The corneal epithelium becomes disorganised and irregular, and its transparency is impaired ; but Bowman's membrane remains intact, and therefore superficial corneal ulceration never occurs. There are infiltrations in the walls, and partial obliteration of the vessels in the sclera and corneal margin, also in those of the iris and ciliary body. The invading vessels enter the cornea from the periphery, and are always situated in the deepest layers. In the early stages, on account of the overlying areas of infiltration, they can seldom be seen clearly, but as the vascularisation progresses these areas tend to clear up, and then they become more obvious. The peculiarity about these vessels is that they appear in great numbers and are always of the same length, giving rise to the appearance known as "salmon patch." Always in the severer cases, and frequently in the milder ones, there co-exists an iritis and cyclitis, with deposits on the posterior surface of the cornea, and ciliary injection, associated with pain and tenderness.

The disease occurs most frequently between the ages of nine and sixteen, but may appear in earliest childhood, and later up to about twenty years of age. It is practically always bilateral, the second eye becoming involved at a varying interval after the first. This interval tends to be longer, the older the patient. The prognosis as regards vision must be a guarded one. As the inflammation subsides the vessels become emptied of blood, but the walls remain as a permanent nebula, and there may be a great quantity of fibrous tissue formation which further obstructs vision.

The causation is always congenital syphilis (very rarely, especially in adults, acquired syphilis), and this gives the indication for general treatment. The local treatment consists of hot applications and atropine in the acute stage, and later yellow oxide of mercury to promote the absorption of the opacities.

Squint.

The various affections of the lens in children, such as congenital, polar and lamellar cataracts, as well as the various conditions found in the retina and choroid, are outside the scope of this article ; but some reference must be made to squint, as it is of frequent occurrence, causes much distress to parents, and may have serious psychological effects upon the children as they grow older. They become self-conscious, shy and retiring, and are apt to acquire an inferiority complex, which may make life very difficult for them. One form of paralytic squint, that associated with damage to the sixth nerve, has already been mentioned, but it is only proposed to deal here with the ordinary concomitant squint of childhood.

The ætiology of squint is still a matter of dispute, but for practical purposes it can be considered to be the result of some anomaly of refraction so pronounced as to prevent the establishment of fusion. This presupposes an error large enough to upset the balance between convergence and accommodation, or a difference in the refractive error of the two eyes sufficiently great to make the visual acuity of one eye much less than that of the other. In these cases, in the interests of clear vision, the image of one eye will be suppressed. This eye will then become amblyopic, or, if the squint develops in very early life, will never learn to see. Squints usually develop in early life, and as most children are hypermetropic, the convergent impulse associated with the

required accommodation tends to make these defective eyes assume a convergent position. The age of onset varies, and in general it may be said that the higher the hypermetropia, or the greater the difference between the two eyes, the earlier the squint will appear. It may occur as early as six months, or as late as three, or even four years of age. It never arises after five, because by that time binocular vision is well established in eyes that have remained parallel up to that age. The indications for treatment are obvious: the fullest correction of the refractive error, care being taken to correct the astigmatism accurately and to ensure that the convergent eye takes part in the act of vision. To ensure this it may be necessary to occlude the second eye for a certain number of hours daily. In older children, from five to six, it will be advantageous to train the fusion faculty by means of some form of stereoscopic exercises. By careful attention to detail it is generally possible to obtain a complete cure both functional and cosmetic. In those cases where this treatment has not been carried out from the earliest moment, operation will give a cosmetically good result, but the functional result will not be so satisfactory.

To conclude this article, it will be well to mention perforating injuries of the globe. These occur very frequently, and their importance is not always recognised. Any injury to the eye should be examined carefully to make sure that the iris is in place, and that the anterior chamber is of normal depth. Any prolapse of the iris or displacement of the pupil should receive prompt and skilled attention, as there exists danger of deep infection of the globe with its attendant possibility of sympathetic ophthalmitis, even from the slightest injury, such as perforation with a needle. The dangers are many, and early treatment generally obviates the more serious consequences of such mishaps.

CHAPTER XLVII.

THE COMMON SKIN DISEASES OF CHILDHOOD

BY

M. SYDNEY THOMSON, M.A., M.D., F.R.C.P., F.R.S.E.

Physician for Diseases of the Skin, King's College Hospital and Belgrave Hospital for Children ; Consulting Dermatologist to the Ministry of Pensions and the London County Council.

It is surprising how the great majority of children's dermatoses fall into one of the four following groups : those due to sepsis or parasites, eczemas and lichen urticatus. Occasionally there is seen a case of psoriasis, ichthyosis, lupus vulgaris, syphilis, etc., but such examples are always comparatively rare, and comprise no more than about six per cent. of a children's clinic.

Sepsis.

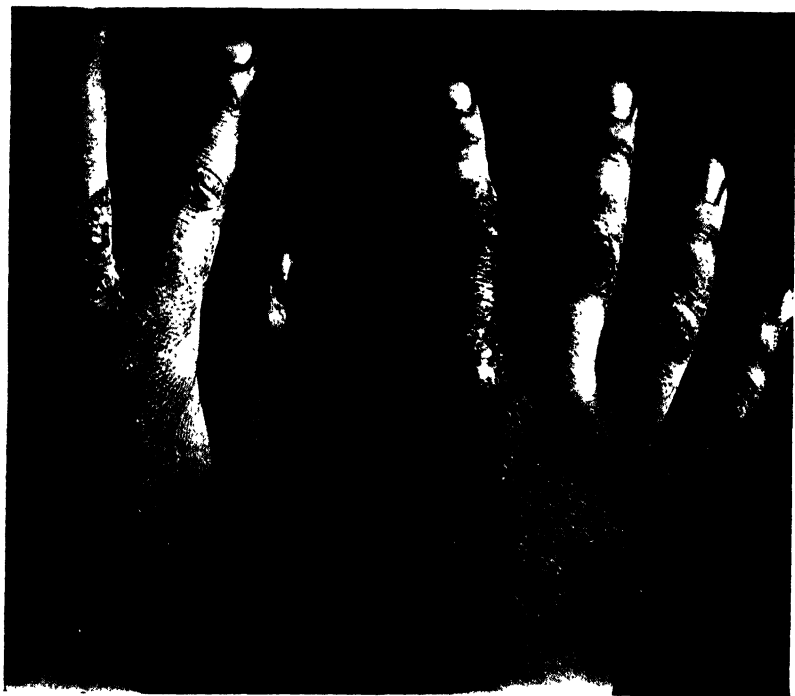
Of the four groups, however, by far the most important is sepsis. It is important not only because the pyogenic infections themselves spread rapidly, and are highly contagious, but also because every itching eruption is liable to become secondarily infected with these organisms. This is easy to understand when we realise that young children have not yet had the opportunity to attain a high degree of immunity to these cocci, that the epithelium and horny layer is still relatively thin and easily damaged, and that the scratch reflex is so primitive that the least irritation immediately gives rise to a scratch reaction. Add to these factors the "normal" grubbiness of a child's hands and nails, and we have the stage set for the appearance of sepsis. Sepsis might, indeed, be likened to the chorus of a musical comedy, always liable to erupt on to the stage and distract attention from the one or two stars present. Yet it is these stars who really matter. In the same way the essential underlying disease may be masked by the sepsis. To put it in another way, one's attention is diverted from

the picture because of the large ornate and golden frame of impetigo crusts. Therefore, although such direct inoculations as scum-pox and gravel-rash are by no means rare, all septic eruptions in children must be most carefully examined before we remain satisfied that no underlying disease is present. This is particularly true in widespread sepsis, where scabies or lichen urticatus may be exceedingly difficult to detect.

Treatment.—Certain generalisations can also be made in regard to treatment. As the horny layer of the epithelium is only thin, local applications should tend to be on the weak side. Any chemical strong enough to kill a coccal spore is strong enough to burn the skin. Our antiseptics should only be strong enough to make the organisms thoroughly unhappy and disinclined to produce large numbers of offspring. The body itself can then be safely left to deal with the original invaders. The dilute mercurial ointments, or even such lotions as that of hydrarg. nigra, are efficient in most cases. But to be efficient they must be brought into contact with the organisms: *i.e.*, crusts, flakes of loose horny layer, etc., must be removed thoroughly, and daily, before the remedy is applied. Although such careful dressing is not very painful, it requires a strong will on the part of the mother to deal truly and well with the lesions on a fractious or crying child. Very often the mother fails, and that is quite understandable, but it is in such circumstances that one usually finds the widespread and desperate cases. A great deal of trouble can be saved to everyone if such children are put in the hands of a visiting nurse for a week. Occasionally we do meet intractable examples in which the children seem to have no power of acquiring resistance, such children being usually somewhat sallow in appearance. Then general tonics such as tinct. ferri perchlor. internally, and general carbon-arc baths, are invaluable.

Parasites.

Pediculosis corporis is to-day much less common than it used to be, partly because of the educational work carried out by the welfare centres and kindred organisations, and to a lesser extent because a social stigma now attaches to its presence which was absent in pre-War days. Nevertheless occasional cases are seen and may be overlooked on account of the estimable maternal habit of dressing the child in clean clothing before coming for treatment. Although one is thus prevented from discovering ova, etc., the



Scabies masked by a secondary eczema. From MacKenna's
"Diseases of the Skin" (*By permission of the publishers*).

THE COMMON SKIN DISEASES OF CHILDHOOD," by M. Sydney Thomson,
M.A., M.D., F.R.C.P., F.R.S.E

eruption itself is usually sufficiently characteristic. Over the upper part of the back there is an indefinite pigmentation with a few small blood scabs and many excoriations. Local treatment with sulphur and sterilisation of the clothing quickly remedy the disease.

Pediculosis capitis is usually only noticed after it has given rise to secondary impetigo of the scalp and face. This disease is also comparatively rare to-day owing to the modern fashion of short hair, with its consequent easier and more frequent toilet. There is a simple and very efficient method of treating these cases. To one pint of 5 per cent. carbolic acid lotion there is added one pint of boiling water. The child is laid on a bed with the head projecting over the edge, a basin being placed on a chair beneath. For ten minutes by the clock the warm lotion is then baled from the basin over the hair, care being taken to ensure perfect saturation at the nape of the neck and immediately above the ears. An old towel is then wrung out in the lotion and the hair tied up in this like a turban. After one hour's steaming in this way the head is simply dried. The carbolic, which is the only simple chemical to penetrate chitin, kills every nit. The whole disease is thus dealt with at once, and there is no need to apply repeated remedies to kill the pediculi as they hatch out, for only by such constant repetition can sassafras, etc., prove efficient. The nits can, of course, later be removed at leisure with a fine-tooth comb washed in 4 per cent. acetic acid.

Pediculosis pubis is only of importance here in that the parasites are sometimes found clinging to the eyelashes, whence they have to be detached by means of forceps.

Scabies was, of course, very prevalent in 1919 and 1920, after which it became increasingly rare until twelve months ago, since when there has been a marked recrudescence in its incidence. In children, secondary infection is always present, and often leads to confusion. But if we remember that the disease may also affect the head and neck in infants under the age of two years, and if we make a habit of being certain that a "run" is seen, mistakes will be avoided. Actually, I think it is important to be able to extract an acaris from its burrow, despite the fact that many authorities regard it as a waste of time. A very little practice soon enables one to become expert, and the constant effort to repeat the success does undoubtedly keep one's powers of observation more acute. This is important, as the pressure of work often

tends to make methods more careless than one would care to confess on cross-examination. Also it must be remembered that patients do very definitely resent being told that they or their children are suffering from the "itch." To be able to demonstrate the acaris under the microscope is both so convincing and interesting that a permanent friend is made of a patient who would otherwise depart in high dudgeon. In infants, the "runs" can often be most easily found on the feet, but in very septic or partially treated cases one may be forced to examine the mother. Often I have been able to find the disease in a parent who seemed to be immune to its effects in that she complained of no irritation at all.

Treatment.—Treatment usually involves treatment of other members of the family if relapses are to be avoided. For the same reason, sterilisation of the clothing and bed linen is essential. Where it is inadvisable or impossible to invoke the aid of the public health authorities, this can be most efficiently carried out by ironing all the materials with an iron so hot that it only just fails to scorch the article. Whatever form of local medication is used, it is important to realise that sulphur dermatitis is easily excited in childhood. Therefore irritation is often present at the end of the course, and the patient must be sufficiently under control to avoid further aggravation by a repetition of the sulphur. Actually the risk of this secondary dermatitis can be lessened by adding 5 per cent. of cyllin to the sulphur ointment.

Acarine Mange.—Sometimes the acarine mange of cats and dogs is communicated to man. The eruption is then exactly like that of scabies, except for the facts that no "runs" are present and the head is often involved, even in adults. Veterinary surgeons see more of this disease than does the physician, under whose care cases rarely come, as the malady dies out rapidly after the removal of the offending pet.

Ringworm of the Glabrous Skin.—Ringworm of the glabrous skin is always typical in that close examination of the spreading edge reveals that it is really a necklace of tiny vesicles. Any antiseptic will cure it rapidly, but in order to prevent relapse it should be continued for a week after apparent cure. From the clinical aspect we can distinguish three types. The most common is that frequently seen on the neck and shoulders of patients suffering from a scalp infection. These rings are pale-pink, indefinite in completeness of outline, and tend to spontaneous cure. When

the rings are larger, redder, and attain the size of a florin, the causative fungus is often animal in origin. Again the domestic pets may prove to be the infecting agents. Lastly, when the rings are very large and have a very marked inflammatory and pustular reaction, an ectothrix or cattle fungus is to be suspected.

Ringworm of the Scalp.—Ringworm of the scalp is one of the vanishing diseases. Although not infrequently seen, out-patient departments are not to-day flooded out with such cases. For this decrease the municipal X-ray treatment centres are undoubtedly responsible. Many errors will be avoided if one acts on the axiom that "all scalp diseases of children are ringworm until that possibility is disproved." The stumps in the partially bald patch are typical in that they are short, twisted, bent, friable, and have lost all elasticity. By the time one has learnt to recognise such a stump, the use of the microscope becomes confirmatory only. In fact, these stumps must be recognised, for the blind plucking of hairs for microscopy will only result in negative reports, as only the normal hair will be picked out. This because the normal hair is longer, and does not snap as do the fungus-riddled stumps. The inflammatory type (kerion) is due to the ectothrix fungus, and is self-limited, as the œdema, etc., causes the infected hair to fall out.

Treatment.—Treatment tries to imitate this effect, and so is devised to remove the infected hair. Anti-parasitic remedies cannot penetrate the hair and follow the fungus down to the root. Therefore the hair itself has to be removed. This is done either by exciting an inflammatory reaction by means of irritating preparations, or by trying to convert the case into one of alopecia by the use of thallium or the X-rays. X-rays act only by making the hair drop, and do not themselves exert any effect whatever on the fungus. It is important to stress this fact, as quite frequently one receives a request to X-ray ringworm of the glabrous skin, an entirely pointless proceeding. Equally it must be remembered that scalp infections do not occur after the age of adolescence. Although X-ray and thallium treatments are carried out extensively and successfully, I feel every sympathy with the parent who refuses consent to such methods. Ringworm is a nuisance, and interferes with education, but it is never a fatal malady. Therefore it is questionable whether one is always justified in risking an X-ray burn, with its resultant permanent baldness, or a therapeutic

misadventure with thallium. It is admitted that such risks are very, very small, but they are present. The irritant ointments are painful, and require careful supervision, but most cases do react satisfactorily to such treatment provided that it is efficiently carried out.

Traumatic Dermatitis and Eczema.

Traumatic dermatitis and eczema may be discussed together, for the line of distinction between the two is vague, and, indeed, many traumatic eruptions are actually eczematous; *e.g.*, in adults we speak of fur dermatitis when it would be really correct clinically and histologically to speak of an eczema consequent on contact with certain dyed furs. Whilst it is true that such a simple external irritant as the primula can produce the eruption, yet even here we have the internal factor of idiosyncrasy; were it not present every individual who ever came in contact with a primula would get eczema. "Eczema" is only a clinical term, and denotes a type of reaction to insult. It is characterised by tiny pin's-head-sized papules or vesicles which individually never increase in size, but which run together to form sheets. These sheets are not clearly defined, but merge gradually into the surrounding normal skin. Certain patients react in this way far more easily than do others, *i.e.*, to lesser trauma or to an agent which is not irritant to the normal person. This "eczematous diathesis," to use an unfashionable but very useful phrase, is a very real condition, and may often be seen very early in childhood.

To take a simple example, most infants get infected with scurf during the first few months of life. In most cases this remains as a simple infection, but in certain patients the local irritation gives rise to an eczematous reaction, starting on the scalp, thence flowing down over the forehead and on to the face like a waterfall. Soaps which contain crude carbolic derivatives or excess of soda are liable to cause eczematous discs of crêpe-like scaling on the face. The same appearance is sometimes seen wrapped round the mouth, and may then be due to a dentifrice or to constant slobbering on the pillow at night as a result of enlarged tonsils and adenoids. Frequently the hands are not perfectly dried, so that chapping occurs between the fingers, round the wrist or over the dorsal surface of the first metacarpal. The irritation of the fissuring and the low-grade secondary infection then give rise to a surrounding



Kerion. From MacKenna's " Diseases of the Skin "
(By permission of the Publishers).

THE COMMON SKIN DISEASES OF CHILDHOOD," by M. Sydney Thomson,
M.A., M.D., F.R.C.P., F.R.S.E.

eczema. Intertrigo may start from the decomposition of locked-up secretions, and so it forms a nucleus round which eczema appears. Sun and cold winds burn and parch the prominences of the cheeks and forehead to cause the typical red patches of traumatic eczema, particularly in fat infants, the victims of carbohydrate overfeeding. In the more severe examples, eczema spreads rapidly all over the body, either from one of these foci or spontaneously. These children in particular have a bad allergic family history. When the disease recedes it often leaves the chronic, thickened and fissured patches seen in the flexures of the knees and elbows; sometimes, however, the trouble starts in these areas.

Treatment.—Unfortunately biochemical, protein tests, etc., are usually valueless, and treatment is restricted to obvious general health regulations, the removal of toxic foci, and local soothing applications. Of these, 2 per cent. of calomel in a zinc cream, or 4 per cent. of ichthyol and 2 per cent. of camphor in the same base, often prove useful. Lassar's paste diluted with an equal part of soft paraffin is also valuable. Soap and even plain water should be avoided, as they are irritant to exposed living cells. Ol. olivæ or ol. lini will keep the affected areas clean. Bromides are often necessary in order to depress the scratch reflex. Splints are of some value, but usually the child will contrive to rub its head or body on the pillow. Treatment is often very prolonged, but patience will produce good results in even the most widespread and chronic cases. For these, some help is sometimes obtained from the use of protein shock, *e.g.*, with autohæmotherapy, milk or hog-spleen injections. But every case invokes very careful supervision and regulation: there is no short cut to the efficient treatment of eczema.

Lichen Urticatus.

Lichen urticatus provides us with a very large group of patients. Here again we have an affection which seems particularly prone to attack the plump child who is so often regarded as a model of perfect health. Such are often the victims of overfeeding, excess of carbohydrates being given either proportionately or quantitatively. But an equally important factor is an excess of fruit, a mistake more common after infancy. Often an outbreak can be determined at will by the administration of chocolate, porridge, bananas, apples, etc., whether fresh or cooked, and it is essential that all such foods should be forbidden during the period of active

eruption. This question of fruit is most important, and although the administration of *syrupus calcii lactophosphatis* (dram 1, t.d.s., p.c.) will help to remove the trouble speedily, constant relapses are bound to occur unless the fruit is incriminated. Recently doubt has been cast on this dietetic factor because it is difficult to reproduce the disease in hospital. Nevertheless, whatever the explanation of this difficulty may be, I am convinced that excess of fruit or carbohydrates is responsible for four out of five cases. Correction of the diet in this respect time and time again produces effects which can only be described as miraculous; this too, without interfering with the environment or normal régime of the child in any way. But the condition is also very common after the acute specific fevers, particularly measles, and in some rachitic children. In fact the older text-books give lichen urticatus as one of the signs of rickets. In other cases frequent relapses seem to be the result of chronic constipation. Certain patients exhibit the disease during the eruption of successive teeth, and these attacks would seem to be the result of a reflex gastro-intestinal disturbance, for alimentary symptoms are often present, whilst the whole may be ameliorated by giving bromides. In some intractable cases threadworms are found, whilst in a few the cause is completely hidden, and may possibly be found in a protein dysmetabolism. The chronic course run by many cases is, however, most often the result of parental carelessness. But although the intestinal condition is of outstanding importance, it is also true that the eruption appears more readily in children who are the victims of overclothing. Such weighty and fashionable oppressions as coarse flannel vests and innumerable layers of coats do undoubtedly play their part.

Whilst this brief account only deals somewhat cursorily with a few dermatoses, their relative frequency is so great that practically all children suffer from one or the other of them at some period of their lives. The general practitioner is therefore bound to meet them almost daily, and for that reason they are worthy of more attention than is usually conveyed by the vague "try white precipitate."

CHAPTER XLVIII.

MODERN METHODS OF EXPOSING THE BRAIN AND ITS MENINGES

BY

LAMBERT ROGERS, M.Sc., F.R.C.S., F.A.C.S.

Assistant Director of the Surgical Unit, The Welsh National School of
Medicine, The Royal Infirmary, Cardiff.

WITHIN recent years there have been radical changes in the surgery of access to the brain and the meninges. Advances have taken place both in the manner in which exposure is effected and in the technique required to bring this about.

A comparatively short time ago certain pages of the standard text-books of surgery were devoted to intracranial topography, and the student had to learn various methods of ascertaining the precise location of the points at which to trephine the skull in order to deal with particular lesions of the underlying brain and its meninges. To-day such expressions as "Chiene's lines," "Reid's base line," or even "trephining the skull," have very little significance, and are reminiscent of times when it was customary to make small openings in the skull at precise points beneath which certain parts of the brain were regarded as lying. These were times when intracranial surgery was in a primitive state, and the mortality from most brain operations was exceedingly high.

One has heard of a small trephine opening being made in a case in which the intracranial tension was considerably raised. When the dura mater was opened, brain substance extruded from the small opening in the bone—to use the expression of a distinguished anaesthetist who was present at the operation—"like tooth-paste out of a tube." Such tragedies do not occur to-day, because small openings such as this are not made in the presence of high intracranial tension. A reduction in tension is first brought

about, and then either the brain is explored through a large opening made by cutting a bone flap which, attached to the overlying soft parts, can be swung away from the dura mater; or else in certain positions, *e.g.*, beneath the temporal muscle or the suboccipital muscle masses, where it may be desired to create a permanent bony defect in the skull, a relatively large area of bone is removed piecemeal with nibbling forceps.

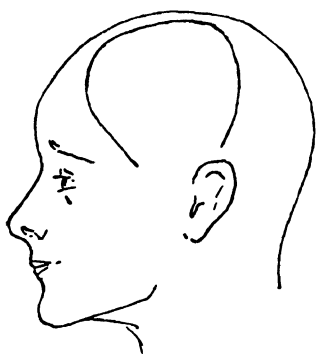


FIG. 1.—Diagram of flap cut to expose the parieto-temporal region of the brain.

Exploratory osteoplastic craniotomy is an operation as necessary and resting upon as sound a basis to-day as exploratory laparotomy, and in the hands of a trained neurological surgeon fraught with as little risk to the patient.

The large osteoplastic flaps which are now in use enable large areas of the brain to be examined with much less risk to the patient than that associated with the production of small trephine openings at precise points on the surface of the cranium. Figs. 1 and 2 show the types of flap which are fashioned to enable the surgeon to gain access to the parietal and temporal, frontal and occipital regions of the brain respectively. It will be seen that these flaps all overlie various parts of the cerebral hemispheres, and that the regions to which access is gained are all supratentorial in position. It will be noticed also that the flaps are designed so that their bases lie towards

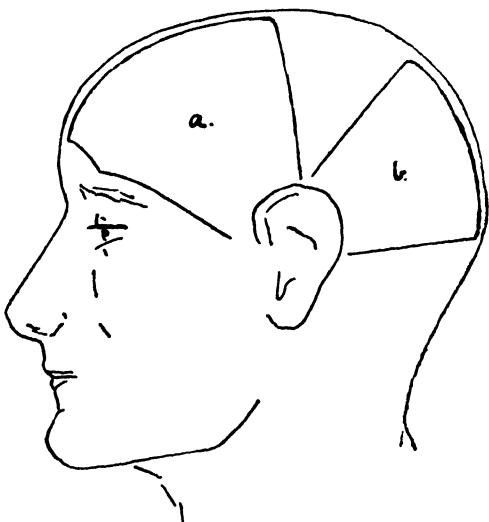


FIG. 2.—Diagrams of flaps cut to expose (a) frontal, (b) occipital regions of the cerebral hemispheres.

the temporal region, *i.e.*, beneath the fibres of the temporal muscle. This arrangement enables the surgeon, should he so desire, to produce a subtemporal bone defect by nibbling out bone in this position, the chief indication for such a procedure being persistent increase in intracranial tension, *e.g.*, because of inability to find or remove a tumour. In such circumstances, namely, persistent increase in intracranial tension, a cerebral hernia will occur here, but this will be well protected by the overlying temporal muscle and other soft parts. With the appearance of the hernia the intracranial tension will be relieved, and although the lesion itself may not have been dealt with, palliation of symptoms and preservation of vision will result, while a second operation may possibly enable the lesion itself to be dealt with effectively.

Although frequently looked upon with much disfavour by members of the nursing staff, and as a rule by the patient's relatives and friends, a cerebral hernia may be highly desirable, the increased tension of the cranial contents which it represents being better expressed extracranially than intracranially, since the relief in intracranial tension may save vision, at least temporarily, and result in a palliation of symptoms.

In cases in which a tumour is successfully removed, or in which after exploration there is no increase in intracranial tension, the bone flap is completely replaced, and there is subsequently no bony defect in the skull whatsoever, because even the small trephine discs removed to enable the flap to be cut can be replaced along the periphery of the flap. Such a procedure is often possible in cases of pituitary tumour.

Very occasionally a blind subtemporal decompression is performed in cases where, although the lesion is obviously supratentorial, the patient's condition is very poor. With wider experi-

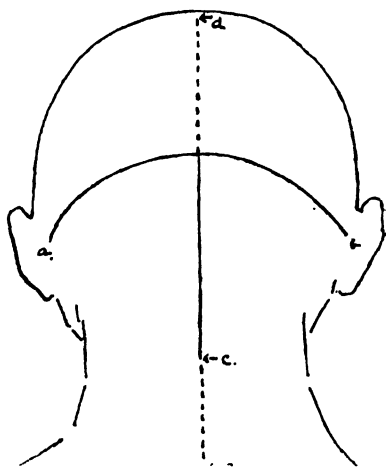


FIG. 3.—Incisions used for suboccipital exposure; *a, b, c*, Professor Harvey Cushing's "cross-bow" incision; *d, e*, longitudinal incision.

ence, however, this operation is becoming less and less frequent, and *the exploratory flap operation with its base in the temporal region remains the procedure of choice in the great majority of cases in which the skull has to be opened for a supratentorial lesion.*

Flap operations have been performed in the suboccipital area, but in this position the writer believes that it is better to produce a permanent bony defect, since such a defect is well protected by the massive suboccipital musculature, and in the majority of these cases a permanent medullary decompression is desirable. Either a longitudinal incision extending from the vertex of the skull to the spinous process of the seventh cervical vertebra is made, Fig. 3 (*d*) and (*e*) (this is all that is necessary in children and in most adults), or Cushing's cross-bow incision is employed, Fig. 3 (*a*), (*b*) and (*c*). In either case bone is removed as far forwards as the back of the foramen magnum, and the opening extended into the posterior and lateral margins of the foramen. As a rule also the posterior arch of the atlas is taken away, while not infrequently laminectomy of the axis is carried out as well. In order to avoid medullary compression, for example, in removing a tumour of the fourth ventricle, or one from the cerebello-pontine angle, a wide bone defect is essential.

Frequently in these subtentorial cases a pressure cone consisting of the cerebellar tonsils overlying the lower part of the medulla oblongata and the upper part of the spinal cord has been forced through the foramen magnum. Only by carrying out such an extensive removal of bone as that described can the pressure be relieved on this cone, in which rests the distorted floor of the fourth ventricle and the important "vital centres" which lie beneath it. The necessity to protect these centres from damage is obvious. In some cases in which, from pressure upon them, respiratory failure has occurred, emergency operations designed to relieve the tension in this position have been successfully undertaken.*

Osteoplastic craniotomy was first performed in 1889 by Wilhelm Wagner, of Königshütte, who used a hammer and chisel for its performance. Many instruments have been devised for carrying out the operation, but that most commonly used has been the wire saw introduced by Leonardo Gigli, of Florence. By means of a special introducer, which is at the same time a guard for the dura

* "The Posterior Fossa Compression Syndrome." Lambert Rogers: *Brit. Med. Journal*, 1933, 2, 100.

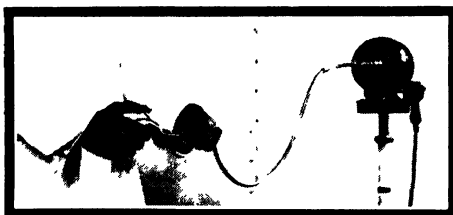


FIG. 4 - Photograph of the skull plough and electro-motor and lead driving it. The method of holding and controlling the instrument is shown

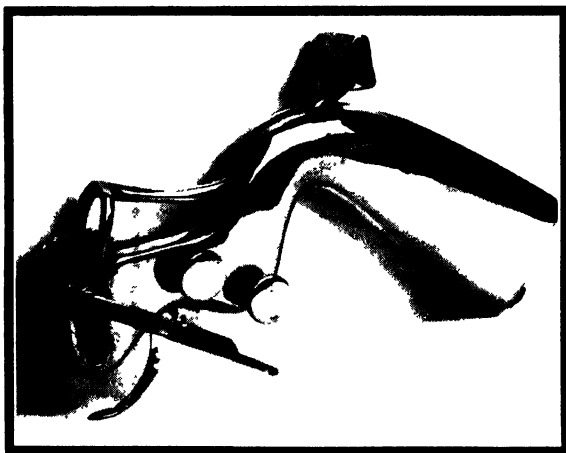


FIG. 5 "Close-up" view of skull plough.

MODERN METHODS OF EXPOSING THE BRAIN AND ITS MENINGES," by
Lambert Rogers, M Sc , F.R.C.S., F A C S.

mater, the wire is passed between small trephine or burr holes, and the bone is then divided from within outwards.

In 1930, with the help of the well-known firm of surgical instrument manufacturers, Messrs. Mayer and Phelps, the writer produced an electrically-driven saw capable of accurately controlled biplanar motion, and so constructed that adequate protection was given to the dura mater, while the guard which enabled this to be done also acted as a separator to free dura mater which might be adherent to the inner table of the skull. This instrument, known as the skull plough (Figs. 4 and 5), has proved successful, and appears to have advantages over Gigli saws, since no special introducing mechanism is required, and the well-known tendency of the wires to break is avoided. The bone is rapidly cut without damaging either the dura mater or the underlying brain. As fewer burr or trephine holes are required when fashioning a flap, this can be more rapidly cut. Holes in the bone are not required at the base of the flap, and it has been found that the base can be broken more easily and accurately between saw cuts than when holes are required, so that this instrument facilitates the production of the osteoplastic flaps illustrated in Figs. 1 and 2.

The skull plough has now been used in this clinic on many occasions, and has proved a very satisfactory type of craniotome. In practice also it has been found to possess a still further advantage over Gigli saws in cases in which the bone is very vascular, because as fast as this is cut with the saw, the saw-cut may be filled with Horsley's bone-wax, and the bleeding thereby effectively prevented.

In the case of suboccipital operations, flap operations being less desirable than the production of a permanent decompressive bone defect in this position, craniotomes such as the skull plough are not necessary to produce this defect, a burr or trephine and nibbling forceps being all that is required. Bone openings are made on either side of the mid-line, and these are connected with each other, and extended by nibbling away pieces of their margins with such forceps as those of Hoffmann or Trotter.

From this brief account it will be readily apparent that within the past few years radical changes have taken place in the methods of exposing the brain and the meninges. Although much larger openings in the skull are now made to obtain access to such lesions as tumours, and the Wagnerian or flap operation is a much more extensive surgical procedure than merely trephining the skull,

the risks to the patient are considerably lessened, and the results of modern cerebral surgery are very considerably improved. Such improvement is due not only to advances in technique, but also to a better understanding of the significance of the state of intracranial tension.

REFERENCES.

Rogers, Lambert: "Osteoplastic Craniotomy: A New Technique for the Resection and Turning Down of Bone Flaps from the Skull." *British Journal of Surgery*, Vol. XVIII, No. 70, 1930, p. 272.

Rogers, Lambert: "History of Craniotomy: An Account of the Methods which have been Practised and the Instruments Used for Opening the Human Skull During Life." *Annals of Medical History*, new series, Vol. II, No. 5, 1930, p. 495.

Rogers, Lambert: "Manner in which Flaps of Various Materials Fracture Along their Bases." *Nature*, November 15th, 1930.

CHAPTER XLIX.

THE TREATMENT OF ACUTE ABDOMINAL CONDITIONS IN CHILDREN

BY

R. A. RAMSAY, M.Ch., F.R.C.S.

Senior Surgeon to the Metropolitan Hospital and to the Belgrave Hospital
for Children; Surgical Consultant, L.C.C. Hospital Service.

General Considerations.

ACUTE abdominal conditions occurring in children show certain differences when compared with those found in adult life, and these differences are not merely of academic interest, but have an important bearing upon the way in which such cases should be handled.

Firstly, a child is relatively undeveloped and unstable, and as such shows, in the presence of any serious illness, great constitutional disturbance at an early period, and also rapidly increasing deterioration; but a child is also young and growing, and therefore has great powers of recovery from the effects of disease if the cause is removed before these effects have become too profound. Secondly, in children, certain abdominal conditions are nearly unknown, such as new growths, ulcers and perforations of the stomach and duodenum, and gall-stones; while, on the other hand, intussusception is almost confined to infants, and pneumococcal and streptococcal peritonitis occur with much greater frequency in children than in adults. And, lastly, the course of acute abdominal disease in children is very rapid, the patients become dangerously ill in a very short time, and may pass into a hopeless condition and die much more quickly than an adult; but if relieved early, unless the disease be general rather than abdominal, the recovery may be also more rapid than in an older patient. If these points are considered,

it is seen that in acute abdominal conditions early diagnosis and early treatment are, if possible, of even greater importance in the case of a child than in that of an adult.

Causes of Acute Abdominal Conditions in Children.

The abdominal conditions requiring surgical attention in children are, as has already been pointed out, not quite the same as in adults. New growths, gall-stones, gastric and duodenal ulcers are most uncommon, and therefore their acute complications are practically unknown, and may be dismissed from the subject under consideration. Abdominal injuries in children may, of course, as in adults, cause hæmorrhage or rupture of a hollow viscus with resulting peritonitis. Appendicitis is probably the most common acute abdominal disease of children, and in them it only too often runs a very rapid course, of which the developments cannot be foreseen ; but every surgeon knows that in many cases gangrene, perforation and peritonitis follow each other with surprising rapidity.

Intestinal obstruction in children is rarely due to strangulated hernia ; most herniæ are reducible in the child by taxis if the muscles are relaxed, either by calming the child, by putting him in a suitable position, by immersion in a hot bath, or by anæsthesia ; it must be extremely rare to see a case of true irreducibility, but if such were found it would require operative treatment as in adults, and even so the hernia is usually easily reduced as soon as the inguinal canal is opened, without the need for division of a constricting band. Internal hernia, either by bands following a previous operation, by a Meckel's diverticulum, or into a peritoneal pouch, will require a laparotomy to find and relieve the obstruction. Intussusception is, as is well known, by far the commonest cause of obstruction in babies, and is usually found in healthy boys between the ages of six months and two years. Inflammation in a persisting Meckel's diverticulum is a very rare condition, usually only recognised when the abdomen has been opened.

Pneumococcal and streptococcal peritonitis are fortunately rare conditions, but are seen more frequently in children than in adults. They differ from other forms of peritonitis in that they are part of a general infection, and do not arise from a removable abdominal focus ; it is for this reason that their treatment is so unsatisfactory. The peritonitis due to perforation of a typhoid

ulcer is a serious complication of a general infection, and is, therefore, a most grave condition.

Certain other acute conditions must be borne in mind, and excluded before a diagnosis of abdominal disease requiring surgical treatment is made. Pneumonia or pleurisy may in any patient, but especially in a child, simulate very closely an abdominal condition. Pyelitis may give rise to such great abdominal pain and general prostration as to suggest the need for a laparotomy. Ureteric or vesical calculus produce severe pain, whose nature is usually clear. Severe diarrhoea may be associated with blood and mucus in the stools, and be difficult to distinguish from intussusception, and in this case it is well to remember that in acute entero-colitis there is usually fever, at least in the early stages, while in intussusception a subnormal temperature is almost always present; further, the absence of a tumour makes the diagnosis clear. Intussusception may also be simulated by that rare disease, Henoch's purpura, but the latter has usually its other signs besides blood in the stools. Hirschsprung's disease may appear to be an obstruction requiring surgical relief; but the history of repeated attacks, the enormous distension and the result of an enema, will usually show the true state of affairs.

Symptoms and Signs.

In investigating these cases the history must be taken carefully. The mode of onset may be most deceptive, and must not mislead the surgeon; for example, the writer has seen a case in which the onset was abdominal pain in a small boy who had eaten a considerable quantity of unripe fruit; in twenty-six hours a laparotomy disclosed a completely gangrenous appendix and a large quantity of free pus in the peritoneal cavity. Inquiry must be made as to previous attacks of abdominal pain, since the attack which is serious enough to need surgical advice may have been preceded by slighter upsets which have not been considered of any importance, but which may throw light on the more recent condition.

The signs of an abdominal catastrophe in a child are not always easy to interpret, since they may be simulated by other conditions. The Hippocratic facies is well marked, and appears early. The temperature is not always a reliable guide; children show a rise of temperature out of all proportion to the cause, and some trivial alimentary catarrh may bring about a quite disproportionate

fever ; also chest conditions and pyelitis may produce fever in cases where there is abdominal pain. The pulse of a child tends to become rapid and variable from very slight causes, and it must be observed carefully several times in conjunction with other signs and symptoms. The tongue becomes furred early in all digestive disorders, slight or serious, and the breath is often offensive in proportion. If vomiting is present it is of the greatest importance to note whether it is accompanied by diarrhoea or by constipation, the latter association being much more suggestive than the former of a condition requiring surgical treatment.

The first symptom of which these patients usually complain is pain : this is of two kinds. Usually the pain which is first present is in the nature of a colic ; it is due to spasmodic contractions of the muscular wall of a hollow viscus, and is felt in the region of the umbilicus or all over the abdomen. Such colic may be simply the result of unsuitable food, but in this case it tends to improve after a few hours, and any colic lasting longer demands serious attention. If the cause is some local inflammation or obstruction, the pain tends to abate after a varying period, because the muscle is tired out or because the inflammatory process has spread to it ; if the part has become gangrenous, any toxic absorption that had been going on may cease, since presumably the venous and lymph vessels are thrombosed, and an associated improvement in temperature and pulse may be noted. But it must be stressed that this abatement of symptoms is often a dangerous sign, since it may be the beginning of spread of infection to the peritoneum, or of ulceration or rupture of the paralysed bowel. Should either of these occur peritoneal pain takes the place of colic, and is found to be localised to some particular region of the abdomen, and to be increased by pressure or by movement.

Abdominal examination not only assists in realising the presence of an acute condition, but also gives useful indications as to its site and nature. On inspection, localised or general distension may be noted, due usually to collection of flatus in the intestines, and less frequently to a free pus in the peritoneal cavity. When peritonitis is present the abdominal wall is held partly or completely rigid, and does not show its usual respiratory movements ; this effort to give rest to inflamed surfaces may be so great as to produce exaggeration of the transverse folds of the skin, a sign which has often been observed by the writer. Rigidity is usually first seen

in the lower abdomen, since, at any rate in children, most peritoneal infections have their origin below the umbilicus; an exception must be made in the case of peritonitis due either to the pneumococcus or streptococcus, where the infection is widespread from the start.

On palpation, local resistance and tenderness are usually found in the same situation as the immobility, since spasm of the muscles is for the purpose of preventing the sensitive peritoneal surfaces from moving on each other. In cases where such tender surfaces lie deep in the pelvis, the writer has frequently found that the insertion of the rectus muscle to the os pubis on the affected side is definitely more tense than that of its fellow, and in the absence of more obvious signs this may be of the greatest value. There is rarely sufficient gas liberated for liver dulness to be abolished, and uniform increased resonance is more usually due to distension of the bowel. The presence of a localised tumour, and its size, situation, shape, consistence and mobility, are important, and may indicate the presence of a localised abscess or of an intussusception.

Absence of stools, especially if no flatus is passed, is a very grave sign, and should be confirmed by giving an enema. Blood mixed with mucus is found in the stools when an intussusception is present, and the writer recalls a case of a very small intussusception found at operation three hours after an explosive passage of blood and mucus in a case where the tumour was not palpable. Diarrhoea is usually the sign of a condition requiring medical rather than surgical treatment, but in all classical descriptions of pneumococcal peritonitis loose and frequent stools are said to be characteristic.

Rectal examination in a child causes more discomfort than in an adult, but also gives more information, since the finger can reach well into the main abdominal cavity, and such an examination should not lightly be omitted; in an infant it is advisable to use the little finger. The presence of faeces should be noted; ballooning, with complete emptiness, suggests an obstruction; distended coils of small intestine may be felt as a tensely elastic bulging around the rectum; tenderness more marked on one side than the other, a sign not always easy to be sure of in a child, suggests local pelvic peritonitis; while the presence of a tumour in the pelvis points to an abscess. The apex of an intussusception may in some cases be felt by the examining finger, but if this is the case the diagnosis

is usually already clear. Lastly, it must not be forgotten that a bi-manual examination may reveal the presence of a stone in the bladder.

Finally, in examining a child with abdominal signs and symptoms, the examination of the chest and urine should never be omitted.

Treatment : General Considerations.

When, after consideration of the signs and symptoms, a diagnosis of an acute abdominal condition is made in a child, with few exceptions the treatment must be an operation with the least possible delay. The possible exceptions are irreducible hernia, which, as already stated, is usually found to be reducible with appropriate treatment; and pneumococcal and streptococcal peritonitis, for which in certain circumstances it is perhaps wiser not to operate at once, if at all.

In all other cases it is, on account of the peculiarities of children, dangerous to delay. It is difficult, if not impossible in a child, to foretell the course of the disease; some conditions may become quiescent and recover, but will at a later stage require exploration and removal of the cause so as to prevent recurrence. But while waiting and hoping for such a result, the disease may take the opposite course. The child's general condition may rapidly deteriorate, and without warning pass into so grave a state that, even if then operated upon, his reactive powers will no longer prevail, and while at the worst the patient may die, at best recovery will be more prolonged than would have been the case had immediate steps been taken. If the diagnosis is not clear the patient should be kept under close observation, and careful watch kept for further signs; during such a period of waiting no purge should be given, since it may hasten serious complications, and morphia should be withheld, since it will obscure signs already present and prevent new ones from being observed, so giving a completely false idea of improvement.

In these cases there is usually little time for pre-operative preparation. Unless an enema has to be given to settle the diagnosis, emptying of the bowel should be avoided, since purges are dangerous, and an enema disturbs the patient and deprives him of fluids. Where the patient is in a state of profound prostration, attention must be paid to the maintenance of body heat by warm clothing and hot bottles; the limbs may be bandaged to assist

in preventing shock ; and it is most important that as great a quantity of fluid as possible should be introduced into the body. From the nature of the case, oral and rectal administration are usually contraindicated, but saline may be introduced subcutaneously into the muscles or into the peritoneal cavity, while the value of blood transfusion in an extreme case must not be forgotten.

Operations for these conditions in children must be as short as possible, the manipulations must be gentle, and only what is absolutely necessary must be done ; long operations only add to the unfavourable general state, and so prejudice the result. As little as possible of the abdominal contents should be brought out, and efforts to avoid evisceration should be made.

The anæsthetic requires careful consideration, and depends largely on the opinion of the anæsthetist, who in these cases should be one of large experience. The writer has found open ether, often with induction by ethyl chloride, to be the most satisfactory ; chloroform is not advisable. Local anæsthetic, while without toxic effects, often makes the closure of the abdominal wound difficult and prolonged ; it is, however, quite satisfactory when a simple incision for drainage of a localised abscess is made. Spinal anæsthesia tends to depress rather than to stimulate, and has not, in the writer's experience, been found satisfactory.

The incision will depend upon the diagnosis. If a definite diagnosis of appendicitis has been made the muscle-split of MacBurney gives the best access, and if pus is found and drainage established there is little risk of spread of infection in the abdominal wall, while the subsequent falling together of the splits in the muscular planes renders post-operative hernia a rare occurrence.

In cases where the diagnosis is uncertain, or where it is unlikely that pus will be found, the incision through the rectus sheath should be employed. A small incision for exploration can be made and extended up or down according to the condition found and the exposure required. It is usually made on the right, since most conditions can so be dealt with. But if pus is found and drainage established through this incision there is danger of infection of the rectus sheath, and if such should occur it may spread extensively up and down the sheath and produce a very serious state of affairs.

Regarding drainage, there is some difference of opinion. It is

said that the peritoneum can deal with suppuration if the cause is removed ; but residual abscesses occur in a certain proportion of cases, and their presence prolongs the illness and seriously affects the general condition ; while an operation to drain them is so difficult and dangerous that it is often wise to wait till they are of such size as to be immediately under and fixed to the abdominal wall, and while waiting for this the child may pass into a very serious state. Therefore, if pus is found it is wisest to insert a large drainage tube, in the case of a localised abscess, into its cavity ; and where the infected area is not shut off, to the bottom of the pelvis, where pus is most likely to collect ; or to the kidney region. A drainage tube through a muscle split rarely causes hernia ; adhesions will form, it is true, but they will form after peritoneal infection even if a tube is not inserted, and in any case they tend to diminish rapidly in the course of a few months.

Operative Details.

The details of the operation will, of course, vary with each individual case. If the cause is an injury a careful exploration will have to be made through an incision chosen with regard to the site of the injury and of most marked signs ; the presence of blood, gas or intestinal contents will suggest the nature of the injury, and the source must be found, bearing in mind that there may be more than one. Bleeding is arrested, a leak or rupture sutured, and the surrounding soiled parts sponged clean. In an early case drainage may be omitted, but if infection is established it is wiser to insert a tube.

In appendicitis the muscle-splitting incision is recommended for reasons already given. If the appendix is easily delivered it should be removed. If general peritonitis is present without shutting off of the infected area, the appendix should be removed and drainage established. If the appendix is enclosed by adhesions, so that its delivery would involve rough handling of inflamed tissues, it should be left, and drainage of the infected area should be established. If an abscess cavity is opened the appendix, if easily found and delivered, should be removed and the cavity drained, but no prolonged search should be made, the appendix being left if its removal would involve breaking down the walls of the abscess. When the appendix is left it is found that its removal after three months is not as difficult as might be expected,

since after such an interval the adhesions tend to become much less troublesome. Finally, the abdomen must be closed carefully in its original layers with a gap for the tube if one has been inserted.

Some surgeons advise expectant treatment of appendicitis after forty-eight hours, hoping that the infected area will become shut off or that the attack will subside. Unfortunately, these desirable results by no means always occur, and if one waits the child may, instead of recovering, go down hill and pass into a state in which recovery may be impossible. Whatever may happen in adults, it is an exceedingly dangerous policy not to operate at the earliest possible moment upon a child suffering from acute appendicitis.

In dealing with an acute intestinal obstruction, unless the cause is an irreducible hernia or intussusception, the operation will in the first place be exploratory. If there is the scar of a previous operation it is best to excise this, and so open the abdomen and search for adhesions, which will probably be the cause of the obstruction. If there is no such scar an incision through the right rectus will give a good exposure through which one can look for bands, a Meckel's diverticulum, or internal hernia into a peritoneal pouch, retro-sigmoid, retro-cæcal or retro-duodenal. If the site of obstruction is not easily found it may be traced by following backwards an undistended coil. Any obstructed loop is freed, carefully examined for viability and, if safe, returned to the abdomen. It is in such explorations that one must endeavour to bring out only a small length of gut at a time, and so avoid the extrusion of a large amount of bowel which often tends to occur; should this happen, the distended coils should be covered with hot sponges and returned to the abdomen as quickly as possible.

In hernia, as already pointed out, true irreducibility is fortunately uncommon; reduction will usually take place as a result of taxis, with or without an anæsthetic; should this fail, the loop will usually return to the abdomen as soon as the inguinal canal is opened. In the extremely rare cases where even this does not occur, the sac must be opened, the constriction divided, and the bowel examined and radical cure performed exactly as in an adult. If the hernia is reduced without incision, it is probably best to defer the radical cure for a few days to allow the tissues to recover from their stretching by the hernia and manipulations, thus making the operation easier and more satisfactory.

Intussusception is usually recognised before operation. In such a case it is, if possible, of more importance than in any other acute abdominal condition to operate without any delay, for the prognosis depends on the duration of the intussusception ; patients operated on within twenty-four hours from the onset nearly all recover, while those where more than forty-eight hours have been allowed to elapse show a high proportion in which the intussusception is irreducible or even gangrenous, and a great number of such late cases have a fatal termination. The operation itself should be rapid in view of the serious condition of the patient and the fact that in an infant such an operation is in itself a severe shock. An incision through the right rectus is best, since the intussusception will usually be found to have started in the last few inches of the ileum. The tumour is gently delivered ; this may be difficult, and if it can be partially reduced inside the abdomen, the decrease in its bulk will aid the manipulations. Once on the surface the intussusception is reduced by gently squeezing and pushing the invaginated portion back towards the neck. While this is being done the external peritoneal coat must be supported, since it has a tendency to tear, and if this happens the tears may need to be sewn up, an added incident which prolongs the operation. It will be found that the last inch of invaginated bowel requires more care than the rest, and always comes out with difficulty ; this fact shows the futility of attempting reduction by inflation, a procedure which does not succeed, may give a false sense of safety if the tumour apparently disappears, and so wastes valuable time. Should reduction be impossible, or should the invaginated part of the bowel be gangrenous, resection will have to be performed ; but this operation is of the greatest severity, and is rarely followed by recovery.

There is considerable discussion as to the best treatment for pneumococcal or streptococcal peritonitis, since these are merely local manifestations of general diseases in themselves of the greatest gravity ; therefore there is no local focus to be removed. For this reason many surgeons recommend treatment of the general infection without operative interference. The difficulty, however, is to make so exact a diagnosis as to be certain of the true condition, and to avoid leaving a removable cause, such as a gangrenous appendix, untreated ; for this reason an exploration is done in most cases, and when the true nature of the disease is ascertained

by the absence of a local focus, by the lack of smell from the pus, by the appearance of the pus, which is flaky in pneumococcal and thin and turbid in streptococcal infection, the abdomen is usually drained. On confirmation of the diagnosis by immediate bacteriological examination, serum treatment may be tried, but is not usually successful, and such cases show an extremely high number of fatal results. If the case is obvious as a peritonitis, due to the pneumococcus or streptococcus, and associated with other localisations such as pneumonia or meningitis, general treatment alone should be tried. But, unfortunately, whatever the treatment, the results are very disappointing.

After-Treatment.

Following an operation for an acute abdominal condition in a child, careful after-treatment is necessary. In view of the profound prostration found in such cases, salines, either rectal, subcutaneous or intravenous, should be given, and stimulants may be required. Blood transfusion is of the greatest value, and in infants the intramuscular injection of whole blood has been found of use. The patient should be nursed in the Fowler position if any abdominal infection has been present, so that pus may gravitate to the pelvis, where toxins seem for some reason to be less rapidly absorbed. A careful watch must be kept on the pulse, temperature and respirations, bearing in mind the tendency for variations of these to be exaggerated in children. Finally, the passage of flatus is, in the opinion of the writer, a most favourable sign, and measures to assist it should be taken, such as an enema soon after operation, the administration of small doses of eserine or of very small repeated doses of calomel. Chest complications are not common in children, but the possibility of their occurrence must be borne in mind.

Prognosis.

The prognosis in the cases under consideration is, in the main, good, but there are a few important exceptions. In any case in which too great delay has occurred, the patient is in such an unfavourable condition that the probability of his recovery is greatly diminished; while in a general infection affecting the peritoneum merely as a local manifestation the prognosis is extremely grave, and early operation is apparently of little value in improving the result. But apart from such exceptions, the very great recuperative

power of children shows itself in the rapid recovery from prostration and the high proportion of satisfactory results which is found in the cases with which this paper deals.

Conclusions.

The subject of acute abdominal conditions in children is immense, and in a short discussion it is impossible fully to cover it in all its aspects. An attempt has been made to review the important points in the subject, and while it has been impossible to go into great detail, it is hoped that attention has been called to certain general principles. These may now be briefly summed up. The rapid deterioration which takes place in a child suffering from abdominal disease or injury is always to be remembered ; but to offset this unfavourable characteristic there is the enormous reactive power of the young. And we must attempt to give the latter every possible chance by allowing it to exert its beneficent effect before the unfavourable factor has obtained too firm a hold. Therefore a diagnosis should be made as early as possible. If the diagnosis is accurate, so much the better ; but if not, the presence of an acute abdominal condition as such should be recognised at the earliest possible moment, even if its cause cannot be determined. When such a diagnosis has been made, with certain exceptions an operation should be performed at the earliest possible moment and in the shortest possible time compatible with care and completeness. If these principles are borne in mind and acted upon, it will be found that the treatment of acute abdominal conditions in childhood gives most gratifying results, and is one of the most satisfactory branches of surgery.

CHAPTER L.

FRACTURES OF THE ANKLE-JOINT AND THEIR TREATMENT

BY

GERALDINE BARRY, M.S., F.R.C.S.

Assistant Surgeon, Royal Free Hospital.

THE ankle-joint is formed by the lower ends of the tibia and fibula, which together form a mortise, into which is received the upper articular surface of the astragalus. The mortise itself and the superior surface of the astragalus are not perfectly square, but are wider in front than behind, thus preventing backward displacement of the astragalus on the tibia and fibula in the normal joint. The lower ends of the tibia and fibula are held together by a very strong interosseous ligament, and the capsule of the joint is strengthened internally and externally by powerful accessory ligaments. The movements which take place at the ankle-joint are dorsi and plantar flexion only ; possibly a small amount of lateral movement being obtainable in full plantar flexion.

Fractures at the ankle-joint are produced commonly by a sudden strain being thrown on the joint in a lateral direction ; the most usual one is when the foot is wrenched outwards as in slipping off a kerb, and to this variety of fracture the name of Pott's fracture is applied.

With forcible abduction and eversion of the foot the articular surface of the astragalus is forced up against the lower end of the fibula, which breaks about three inches above the malleolus ; at the same time the strain on the inner side of the joint causes either rupture of the internal lateral ligament or fracture of the internal malleolus. If the violence is still greater the astragalus is forced up between the tibia and fibula with tearing of the interosseous ligament, producing a fracture dislocation of the joint called Dupuytren's fracture. In addition to the injuries already

mentioned in a Pott's fracture, a small piece of bone may be torn off either the front or back of the articular surface of the tibia, and this is one of the causes of difficulty in reduction of the fracture.

Wagstaffe's fracture is the opposite of Pott's fracture, and is produced by forced inversion of the foot, which causes rupture of the external lateral ligament, with fracture of the internal malleolus.

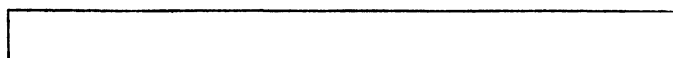
The deformity produced by a Pott's fracture is three-fold : the foot is held abducted and everted, and is displaced backwards ; this is commonly associated with plantar flexion of the foot. The backward displacement is important, and is due to alteration in the shape of the mortise formed by the lower ends of the tibia and fibula, which becomes wider posteriorly due to rupture of ligaments or the wrenching off of a fragment of bone from the posterior lip of the articular surface of the tibia, thus allowing the astragalus to fall backwards.

A certain proportion of these fractures may be compound ; the broken edges of the internal malleolus may pierce the skin, or occasionally the fracture of the fibula may be compound. The injury to the ankle-joint is followed quickly by much swelling and bruising of the surrounding parts ; the œdema appears first, and there is an effusion into the joint which fills up the normal hollows round the joint, and obscures the bony points, and may lead to some difficulty in diagnosing the exact position of the fragments.

Treatment.

The first essential in treatment is to have an X-ray taken of the bones involved ; this is always necessary from a medico-legal point of view, and as an aid to treatment it gives invaluable help by demonstrating the position of the bones and the presence of additional fragments torn off the tibia which cannot be felt clinically. An X-ray should also be taken after reduction to confirm the good position produced by the manipulation. There are several methods of treating fractures round the ankle-joint, the simplest of which is by means of plaster of Paris. By this method the deformity is reduced, and the leg and foot immobilised by fixation in plaster of Paris, which reaches from the toes to the condyles of the tibia. There are certain contraindications to this line of treatment : (a) The fracture may be compound, in which case, if this method is used, it will be necessary to cut a "window" in the plaster opposite the wound to permit of dressings. (b) The degree of swelling

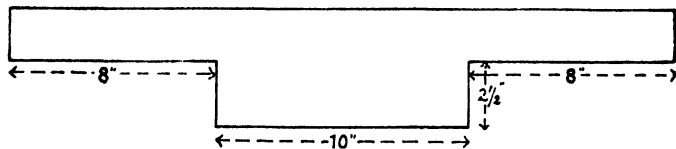
and œdema round the joint may add to the difficulty of applying a plaster ; for instance, if the plaster is applied immediately after the fracture, before the swelling is at its maximum, it may lead to increased tension inside the plaster, with impairment of the venous return and œdema of the toes ; or if the plaster is applied when the swelling is at its greatest, then in the course of a few days the plaster may become too loose. These are difficulties, however, which can be overcome, and for the majority of cases of simple



Upper band : 26×2 in. ; made of 16 layers.



Two side bands : each $26 \times 3\frac{1}{2}$ in. ; of 24 layers.



Lower band : 26×2 in., with heel-piece ; of 16 layers.

FIG. 1.—Diagram to show the composition of the Delbet splint.
(Reproduced by kind permission of the *British Journal of Surgery*.)

Pott's fracture, immobilisation in plaster of Paris will be found a simple and very satisfactory method of treatment.

Method of Reduction and Application of Plaster.—An anæsthetic is usually advisable for reduction of the fracture ; if, however, one is not given, it is most important to procure muscular relaxation, especially of the gastrocnemius muscle, and so the reduction must be performed with the knee flexed. After flexion of the knee, the foot is grasped and manipulated into full inversion and adduction of the back part of the foot, thus forcing the astragalus back into its normal position and restoring the normal plane of the mortise formed by the lower ends of the tibia and fibula (this is the most important part of the manipulation, because a foot which is allowed

to stay in abduction is of necessity a weak one, with an alteration in the weight-bearing angle, and consequent pain and loss of function and the probability of the onset of osteo-arthritis at a later date).

At the same time the foot must be dorsiflexed on the leg to at least a right-angle, and the heel must be pushed well forwards, and a slight amount of inversion of the foot obtained to prevent subsequent flat-foot. When the reduction is complete the foot and leg are then bandaged with cotton-wool, which has previously

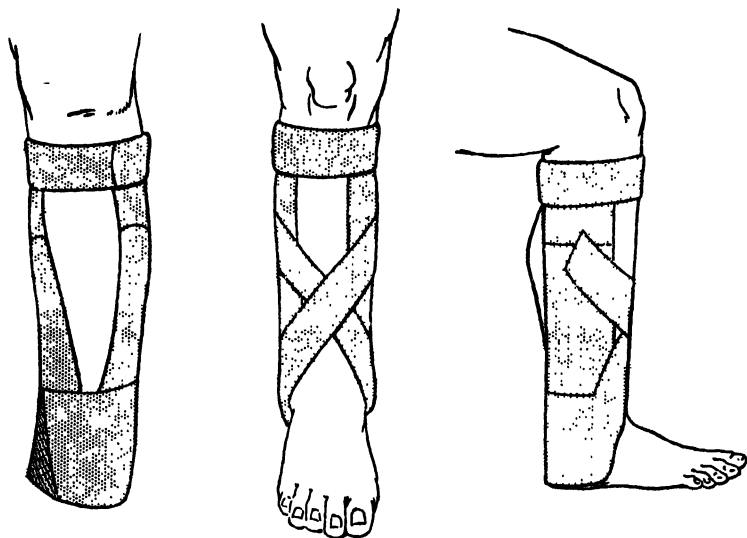


FIG. 2.—Showing the Delbet plaster applied.
(Reproduced by kind permission of the *British Journal of Surgery*.)

been cut into strips and rolled as an ordinary bandage ; then the foot and leg, from the condyles of the tibia to the toes, are encased in a light plaster of Paris splint. This plaster can be left intact for from three to four weeks, and the patient encouraged to walk on it after the first ten days. At the end of three or four weeks the plaster can be discarded altogether.

In dealing with the question of the amount of œdema present, there are three ways of overcoming this difficulty :

(a) Efficient padding under the plaster with soft wool, which relieves the direct pressure of the plaster on the limb and yet enables it to be applied with sufficient firmness to hold the broken fragments. (b) The lapse of one or two days after the fracture

before the application of the plaster to allow the œdema to decrease, at any rate to a slight extent. (c) The immediate "bivalving" of the plaster, *i.e.*, the cutting of the plaster down both sides into anterior and posterior halves after application, so that if the œdema increases the anterior half of the plaster can be slightly lifted off the posterior one, and so give the extra room required.

The advantages of this method of immobilisation in plaster of Paris are: firstly, its simplicity—the plaster is easily applied, and no splint or extension is necessary, the nursing and after-care are reduced to a minimum, and the patient can be treated in his own home under supervision; secondly, the position after reduction is easily maintained, and the deformity does not tend to recur; thirdly, the patient can be encouraged to walk about in the plaster early, and thus keep his muscles exercised and improve the circulation of the limb.

After-treatment is the same in all cases. When the patient begins to walk without the splint he should wear a surgical boot with the sole and heel raised half an inch on the inner side to take any strain off the inner side of the joint and prevent post-traumatic flat-foot. If the fracture has been a more serious one it is often necessary for him to wear a surgical boot with a side-iron reaching up to the condyles of the tibia for two or three months.

Alternative Methods of Treatment.

The Delbet Plaster.—This method of treatment of fractures of the leg by plaster of Paris was originally used in France, and has now become quite popular here. It is an ambulatory method of treatment, and instead of encasing the whole leg and foot in plaster, four strips of muslin steeped in plaster cream are used; of these, two are vertical lateral strips reaching from the tibial condyles to just above the sole of the foot, and the other two grasp the vertical ones at their upper and lower limits, the superior one being applied horizontally in a circular manner round the tibial condyles, and the lower one in a figure-of-eight manner round the malleoli and up the front of the leg. The lateral strips of muslin should be about three inches wide and two feet long, and the upper horizontal one two inches by two feet; and the lower strip twenty-six inches in length, with the central portion forming the heel-piece, ten inches by four to five inches, and the two lateral ends eight inches by two inches each. (See Figs. 1 and 2).

The patient is first anæsthetised, and the fracture reduced and held in the corrected position ; the two horizontal strips are then placed in position and folded over the vertical strips, which are placed on either side of the leg. The upper one is wound firmly round the tibial condyles, grasping the vertical ones as it goes, and the lower one has the heel-piece moulded round the heel ; and then the two side strips continuing the heel-piece are folded in a figure-of-eight manner up the front of the leg, keeping the malleoli in correct position and fixing the lower ends of the vertical strips.

The advantages of this method of treatment are that the plaster is a lighter one than the previous variety, that the patient can walk within a few days of the application of the plaster, and that he is enabled to perform active movements of dorsi-flexion at the ankle-joint at an early date. The plaster may have to be replaced by a tighter one as the œdema subsides, and it needs supervision during the whole time of immobilisation.

Treatment by Traction.

Traction may be required in those cases of fracture round the ankle-joint where there is rupture of the inferior tibio-fibular ligament and subsequent displacement of the astragalus upwards between the tibia and fibula. If the displacement is not easily reduced, or tends to recur after reduction, traction is the best method of producing and maintaining the correct position. Traction may be applied in two ways :

(a) *Bone Traction*.—By means of a transfixion pin or Kirschner's wire through the os calcis. In this method a thin pin or wire is passed through the posterior part of the os calcis behind the plane of the ankle-joint ; to the ends of this pin a stirrup or U-shaped tractor is fixed, and from this stirrup a weight of 8–15 lb. is suspended over a pulley. The weight used is in proportion to the age and muscular development of the patient. The leg is supported in a Thomas' splint, and counter-extension is provided by raising the foot of the bed on blocks. The advantage of passing the pin through the posterior part of the os calcis is that it prevents over-action of the gastrocnemius, and by the direction of its line of pull tends to force the foot into dorsi-flexion. When employing any method of traction it is important to see that the foot is in the correct position of adduction and inversion. The pin can be

removed at the end of three to four weeks, and the leg placed in a "bivalved" plaster splint, and after this the after-treatment will be the same as has been already described.

(b) *Skin Traction*.—Instead of applying traction through the bone direct it can be applied through the skin by means of Sinclair's glue. The composition of this glue is fifty parts each of dry glue and water, and two parts each of glycerine thymol and calcium chloride. The glue is applied warm after the skin has been thoroughly washed, and it can be used to glue strips of muslin or a foot-piece to the sole of the foot, by means of which traction may be applied to the foot as a whole; from this point the procedure is the same as for bone traction. Sinclair's glue can also be used in the form of treatment known as stocking suspension: in this the leg is encased in a cotton stocking which is fixed just below the knee by the glue; the lower end is then slung up to a cradle with the foot in the correct position of adduction, inversion and dorsi-flexion.

Open Operation.

It is sometimes necessary to operate upon fractures round the ankle-joint; there are three chief indications for operation: (a) To remove fragments of bone from the joint which are preventing dorsi-flexion. (b) To clean up the wound in a compound fracture. (c) Occasionally in a Dupuytren's fracture it may be advisable to operate and bolt the lower ends of the tibia and fibula together, if other methods of procuring reduction and fixation of the fragments have failed.

CHAPTER LI.

A PRACTICAL SURVEY OF SPA FACILITIES

BY

G. V. WORTHINGTON, M.B., B.Ch.

Llandrindod Wells.

If the value of spa treatment in the cure or prevention of disease were sufficiently well known, many sudden deaths and many years of suffering and crippledom might be prevented, and many useful lives prolonged in happy content, and our spas would be overcrowded.

Under the head of spa facilities are included situation and climate, hotel and other accommodation, character of the surrounding country, facilities for exercise and amusement, and uses of medicinal waters and baths, and all modern electrical and other physical treatments, including radiotherapy. Special articles have been contributed on the climate, the classification and internal administration of waters and prescribing of baths, and the prescribing of some forms of physical treatment. I therefore propose to give a short description of the baths and some of the treatments found at all well-equipped spas, followed by special facilities found at each individual spa.

For a spa to become a member of the British Spas Federation of this country, it is required that it should possess an active medicinal spring or springs of proved value, and an adequately equipped modern bathing establishment. At a modern spa, therefore, you will find a medicinal spring or springs and bathing establishment equipped with not only medicinal waters, immersion baths and different forms of douches, but also, in most cases, the necessary hot-air, vapour, radiant-heat and all forms of electrical and other physical treatments, also expert masseurs and masseuses, both English and Swedish.

The really essential possession of a spa is its medicinal water or waters: all others, although very necessary, may be considered as accessories. The essence of spa treatment is the elimination of toxins by the drinking of medicinal water and the removal of the result of the actions of these toxins by the various baths and other accessory treatments.

In a short general survey it is impossible to go into theories of origin of springs and their action and that of the various forms of baths. It is, however, important to note, when considering the action of certain group conditions, whether they are hypertonic, isotonic or hypotonic, and accordingly whether they are laxative or purgative, slightly diuretic or markedly diuretic.

There are a few points about medicinal waters which are not generally realised. It is agreed amongst hydrologists that medicinal water cannot be manufactured, and that its analysis may give no very accurate indication of its action. An artificial water, made according to analysis, except possibly in the case of strong purgative waters, does not by any means produce the same effects as the natural spring. The action of a spring cannot accurately be foretold; it must be learnt by experience. The weaker mineralised springs are often much more active than the stronger.

Baths.

The majority of the following baths are found at any modern spa. A short description only will be given of each.

Immersion Baths.—These usually consist of natural medicinal water, given warm (94°–98° F.), hot (98°–104° F.), very hot (104°–110° F.), cold (85°–65° F.), or indifferent (92°–94° F.), according to the action required; and are followed, or not, as considered necessary, by a stimulating douche. They may be full, up to the chin, or three-quarters or half, just above or up to the waist, to limit the hydrostatic pressure. They may be medicated by the addition of extract of pine or other aromatic oils or tar solution. They may be made effervescent with carbon dioxide or air. When effervescent they are much used for certain cardiac conditions at spas where such conditions are especially treated, and are usually administered in connection with special resistance exercises.

Another form of immersion bath is the *pool bath*, sunk below the level of the floor, and having steps down into it, or a special chair or sling, as used at Bath, by which the patient is lowered into it.

The advantage is the extra freedom of movement allowed and the possibility of manipulation by an attendant who can enter the bath. A cool or cold bath with friction is valuable in obesity, the friction allowing the bath to be taken at a lower temperature. Another form is the *aerated sitz bath* found at some spas, and is used, hot or cold, when the action required is to be localised to the lower part of the body, the legs being excluded. *Local immersion baths*, such as foot, leg and arm-baths, are used where a local and not general effect is required.

Sprays.

By a spray is usually meant a stream of water, broken up by a nozzle perforated by several small holes and passing through under a relatively low pressure. It is used where a gentle, soothing impact is required, and may be general or local, and of any temperature, cold or hot, as required. It is used for toning up the local vascular or muscular system and for helping to reduce thickenings and local indurations.

The best example of a general spray is the *circular needle bath*, tonic or sedative, according to the temperature and pressure at which it is given. The temperature may be varied during the bath, and the greater the variation between hot and cold and the higher the pressure, the more stimulating and tonic the effect. It is particularly useful when given after other procedures—such as hot immersion baths, which are likely to be too sedative or enervating—and as a general tonic in case of debility. In cases of secondary anæmia rapid improvement usually takes place under its action.

Douche.

By a douche is usually meant a single stream of water passing through a metal nozzle at a comparatively high pressure. The temperature and pressure are varied according to requirements. The bore of the nozzle is usually circular and may be especially small, when the resulting douche is called a filiform douche, and is used as a counter-irritant. Or the bore may be flat, as in the fan douche, for use when reduced pressure is required for the more delicate parts of the body. In apparatus for giving sprays and douches is fixed a small metal chamber called the mixing box, and a pressure gauge in clock form to enable the attendant to regulate temperature and pressure; the water coming out of the mixing box passes over a thermometer which indicates the temper-

ature. It is most important to watch the pressure and temperature, for these are the two factors which determine what the action of the spray or douche will be and whether the result will be good or bad.

A favourite and particularly valuable form of douche is the *Scotch douche*, which is an alternate long hot and short cold jet douche applied to any particular part of the body required. It is frequently given after the application of heat to reduce swelling, thickening or induration, and to stimulate circulation and tissue metabolism. It is often given, too, on the spinal region after an immersion bath or circular needle spray, and is then one of the most valuable and stimulating of all hydrological procedures.

Douche Massage Baths.—These consist of systematic massage and manipulation under a douche or spray of moderate pressure, the patient either sitting or lying down. The two best known are the Aix and the Vichy baths.

Aix Douche Massage.—In this bath the patient sits on a chair, the attendant performing various massage movements and manipulation under a douche of a temperature and pressure prescribed, starting at the peripheral end of the legs and arms in turn ; working up to the trunk, he follows on to the abdomen, back and chest. Two attendants often work at the same time, thus reducing the duration of the bath.

Vichy Douche Massage is given with the patient relaxed, lying down on a canvas or rubber water-bed, under a series of rose sprays of prescribed temperature. The massage and manipulation is given in the same order as in the Aix bath. Owing to the recumbent position, the abdomen, back and chest are more easily dealt with than in the Aix bath. The Aix bath tends to lower the blood pressure, the Vichy bath to raise it. The Aix is more valuable in dealing with local conditions as found in joints or special groups of muscles ; the Vichy bath is more useful in general treatment. At Buxton the douche is given in the Aix manner, except that the patient lies in a metal trough of warm water. At Harrogate the patient lies down on a Vichy couch while the abdomen, chest and back are being dealt with. In both cases the douche is used, not a spray.

Colon Lavage.

An exceedingly valuable treatment, useful in intestinal toxæmia and fermentation, various forms of colitis, constipation and the many conditions secondary to them, such as arthritis and other

forms of rheumatism. It consists of washing out the large intestine with medicinal or plain water, or normal saline solution, at 100° F. ; to any of the fluids used a weak antiseptic solution such as permanganate of potash, which I have employed for some years past, may be added. It is given by means of a glass container which can be raised or lowered as required, to regulate the pressure. To the bottom of the container a long rubber tube is attached having a metal nozzle fixed on the end ; to the nozzle a rubber rectal tube of from eight to twelve inches long is fitted. The rectal tube is well lubricated and passed for some four or five inches into the rectum, and through it the large bowel is washed out by means of two douches of natural or prepared water ; the first douche is intended to clear out the contents of the bowel, the second to clean the mucous membrane. There should be an interval of from eight to ten minutes between the giving of the first douche and the second.

Vapour Baths.

Local and general vapour baths are of several varieties, and are found at most spas. The Berthollet local steam vapour bath, the general cabinet vapour bath for the whole body, and the Wilde thermal couch, are those most commonly used. The principle is the same in all, and they are valuable when moist heat is required, often starting the action of the skin when dry heat has failed.

Packs of simple bran, mustard, peat or mud (Fango) are all to be found at most modern spas, and form a very useful and simple way of applying moist heat, either locally or generally.

Paraffin Wax, a comparatively new method of applying heat, is used in the form of local baths or packs (sprays), and is very useful in the treatment of stiffened or sprained joints, and for reducing inflammation, and thickening and softening fibrositic deposits in muscles and joints.

Many forms of radiant heat—luminous and non-luminous, local and general—ultra-violet ray and infra-red ray lamps, and all the usual electric treatments, such as ionisation and diathermy, will be found at the modern spa. These treatments will be described in another article.

Arrival and Investigation.

The patient should come armed with as complete a statement by his medical man as possible of the conditions to be dealt with.

He should not come when suffering from any acute condition : acute conditions are not suitable for spa treatment ; nor is it desirable for those suffering from malignant disease. Radiograms and any laboratory reports available should accompany the patient, and inquiries for appropriate accommodation should be made before leaving home. An appointment should be made with the spa physician, either by letter before leaving home, or by telephone immediately on arrival. Defects of teeth, tonsils, nasal sinuses, gall-bladder, prostate or uterus should have received attention, and any discernible septic focus should be eliminated before leaving home.

On arrival of the patient, the spa physician takes a history and makes a complete examination, including an investigation of secretion and excretion if required. On the first occasion the patient should preferably be seen in bed. Special attention is paid to the nasal sinuses, teeth and tonsils, where sepsis is often found, and which are frequently so closely connected with various forms of rheumatic diseases and many forms of digestive disturbances. The chest is examined for evidence of bronchial catarrh and basal congestion, and for any signs of cardiac trouble, which is often present in arthritic disease. The blood pressure is taken, and the abdomen is carefully gone over in order to detect signs of chronic constipation or fermentation, distended cæcum and ascending colon, or dilated stomach, the latter often being apparently a reflex from the former, and disappearing with the clearing up of the fermentation.

Evidence of malignant disease is carefully looked for ; it is not uncommonly found to be the cause of obstinate sciatica. The liver and spleen are examined for enlargement and evidence of congestion. The joints are examined manually for limitation of movement, adhesion, thickening or tender areas around the capsule, bony outgrowth ; by auscultation—a very valuable method—for the finer or coarser crepitations, friction sounds and bony gratings, each of which has its own significance. All the joints should be examined by this method, for not only can the stage of advancement of the disease be estimated, but the total amount of rheumatic invasion can be fairly accurately calculated. It is often found that even when only a single joint is complained of, all the larger joints are infected, and one often finds evidence of considerable rheumatic invasion in a patient who has never complained of any rheumatism at all.

The muscular system is then examined for thickenings and nodules, increase of tone, etc. Fibrositis may be found anywhere where fibrous tissue exists, but the commoner situations are neck, shoulders, upper arms, muscular groups around the elbows, the muscle groups along the spine, especially in the lumbar region, and in the neighbourhood of the iliac crests. Finally, the reflexes are tested. The pain of locomotor ataxia has not infrequently been mistaken for some sort of rheumatism.

The history should be taken carefully, and the following points should be noted : Any history of the past infection of nose, throat or teeth; any liability to bilious attacks, frequent indigestion, appendicitis, prostatic or urinary infection, and operations for malignant disease (especially in cases of lumbago, sciatica and other forms of neuritis). Radiograms, if not available, are taken of teeth, joints, spine, chest and abdomen, if required, and will help to exclude malignant disease in doubtful cases.

Treatment.

Since nearly all chronic disease is the result of some toxic agent, and the aim of spa treatment is to eliminate toxins, unless there is some definite contraindication, almost 100 per cent. of patients are put on prescribed doses of one water or another, or perhaps a combination of waters. The appropriate baths and any other necessary treatment are carefully prescribed by the physician. Directions are given as to diet, exercise and rest, hours of sleep, and many other details, often small in themselves, but necessary if the best results are to be obtained. The patient should be kept under observation during his stay, and treatment must be varied as required. He should be instructed in the way he should go in the immediate future, a report being sent to his medical attendant at home.

Individual Spas.

All spas, with more or less justification, claim to treat most forms of rheumatic disease. The following are the conditions especially amenable to "cure" at individual spas, and the special facilities available at each spa. Elevation and population, both factors to be considered when choosing a spa, are given in brackets. At all modern spas will be found one or more hotels, at the larger spas several, giving first-class accommodation; and in addition there are smaller private hotels, boarding houses and apartments to suit all classes and all pockets. Lists can always be obtained

from the spa manager or town clerk. In all modern spas there are gardens or commons where exercise is taken: tennis and golf for the able-bodied, and other amusements, such as croquet, for those who are unfit for exercise, besides cinemas, theatres and winter gardens.

Bath, Somerset (100 to 600 ft., pop. 68,648).—Our only effective winter spa. Bath is a sedative and typical thermal spa; it has been noted for generations for the treatment of rheumatoid or infective arthritis and other forms of rheumatic disease. A feature is made of the hot pool baths, and under improved technique the treatment of both spastic and flaccid hemiplegia by controlled movements has proved effective. Special attention is being paid to the treatment of spondylitis. The "Bath thermal vapour," both general and local, is a recent development. Bath has a very efficient mineral water hospital of 150 beds for the treatment of poor people, with every laboratory facility. First-class hotel accommodation and every kind of amusement are available.

Buxton, Derbyshire (1,000 ft., pop. 15,651).—This is a very bracing spa, at times bleak; it is subthermal, and is well known for successful treatment of gout and chronic rheumatic disease, including arthritis. A special improved form of douche massage bath has been developed. The most recent improvement is the colloidal peat bath, in which the peat is held in suspension in the medicinal water. The public gardens, concert hall and winter gardens are excellent; and there is a permanent orchestra. Every kind of accommodation is available, including the Devonshire hospital and laboratory.

Bridge of Allan, Scotland (pop. 3,579).—This is a mild, sedative spa, situated on Allan Water. Chloride water is specially useful in catarrhal conditions of digestive or respiratory tracts. First-class hotel accommodation is available.

Cheltenham, Gloucestershire (150 ft., pop. 48,444).—This is a sedative spa, favoured by Anglo-Indians and others who wish to avoid a too bracing climate. It can boast the only true alkaline water in this country. The waters are chloride, sulphated, alkaline and chalybeate, and are specially useful in the treatment of torpid liver, gall-stones, gastro-intestinal catarrh and hyperpiesis. There are extensive gardens and a flat promenade. The Cotswolds are in the neighbourhood. The hotel accommodation is good.

Droitwich, Worcestershire (200 ft., pop. 4,558).—Droitwich is a true brine spa. Brine (nearly saturated) is used with success

in the form of immersion baths and douches for rheumatic diseases generally, including rheumatoid arthritis, and for some scrofulous conditions. A special feature is made of the brine swimming baths, in which, owing to the buoyancy of the brine, re-educational movements are assisted to a marked degree ; these are very helpful in the treatment of stiff joints, cases of fracture, infantile paralysis, and in hemiplegias. Orchestral music, gardens and good accommodation are available.

Harrogate (400 to 600 ft., pop. 38,885).—This is a very bracing, stimulating tonic spa, offering a vigorous cure ; equipment is complete, and the bathing establishments are particularly fine. There are a great variety of springs, and Harrogate is fully justified in claiming to be a general spa. Special features are modifications of the Aix douche and Harrogate peat baths (local peat). Nose and throat irrigation and atomising apparatus for use with natural medicinal waters are available. There are winter gardens and the Stray, every kind of amusement, and a hospital of 150 beds. Harrogate is a training centre for attendants, and grants certificates.

Leamington, Warwickshire (200 ft., pop. 29,000).—This is a sedative spa ; special stress is laid on the medicinal water baths. The water is of the cold sulphurated chloride type, and is anti-catarrhal in action ; it is claimed to be particularly useful in catarrhal conditions of the liver and gastric intestinal tracts. Special attention is being paid to cardio-vascular cases. The Jeafferson Gardens are a feature of the place: they are beautifully kept, and provide an excellent exercising ground on the flat. Two swimming baths are available, and there is a permanent orchestra.

Llandrindod Wells, Wales (750 ft., pop. 4,605).—This is a bracing, tonic, stimulating spa, noted for its mountain climate and the beauty of the surrounding country. The waters are various, the chief of them being saline, isotonic and hypotonic, laxative and diuretic, sulphur hypotonic, markedly diuretic, chalybeate, tonic. These saline springs are anticatarrhal in action, specially useful in gastro-intestinal and biliary catarrh, congested liver and spleen, and the chronic stage and after-effects of tropical diseases. The diuretic waters are valuable in rheumatic disease and catarrhal and infective conditions of the urinary tract. Every kind of accommodation is available, and sports include golf, tennis, croquet and fishing. The common and the Rock park are at hand for exercise ; there is an orchestra during season.

Strathpeffer, Scotland (150 to 300 ft., pop. 875).—This is a typical purely sulphur spa, the original home of peat baths. It is specially useful in chronic rheumatic diseases and gout, also in skin trouble, especially gouty eczema.

Trefriw, Wales (pop. 780).—This is a mildly sedative tonic spa, noted for its extremely strong chalybeate water. The water must be taken in very small doses. A special feature is the export of water in bottles for home treatment of anæmia and debility after acute and exhausting diseases. It has a small bathing establishment.

Woodhall, Lincolnshire (almost sea-level, pop. 1,635).—This is flat and sheltered from cold winds; it is a comparatively bracing spa on the east coast. The waters are saline, containing bromine, iodine and calcium, used internally and externally as baths and douches. It is especially noted for treatment of certain conditions peculiar to women, and for glandular enlargements; in this connection the Woodhall Fango pack is found useful.

Llanwtyd Well, Wales (800 ft., pop. 1,171).—This is not in the Spa Federation. It is noted for a remarkably strong sulphur spring, and claims to treat successfully most rheumatic conditions and some skin diseases. With specialisation in skins, there should be a good future for the spa. The hotel accommodation is good.

CHAPTER LII.

THE ÆTIOLOGY AND TREATMENT OF ANÆMIAS DUE TO DEFICIENCIES

BY

L. S. P. DAVIDSON, B.A., M.D., F.R.C.P.E.

Regius Professor of Medicine, University of Aberdeen.

PREVENTIVE medicine demands a knowledge of the factors which cause disease and of the mechanisms by which they act, while for curative medicine a knowledge is required of the therapeutic agents to be employed, their dosage and the causes which inhibit or delay their action. Treatment must be preceded by accurate diagnosis, and for this a suitable classification is of definite value. The rapid increase of knowledge regarding the anæmias which has occurred during the last ten years necessitates a revised classification, which, to be of practical value, must group the different types of anæmia into divisions, based on a combined study of pathogenesis and therapeutics. The term "primary," as used formerly, must be abolished, since every anæmia is secondary to a cause; thus, hyperchromic macrocytic anæmia (pernicious anæmia, sprue, etc.) and hypochromic microcytic anæmia (post-hæmorrhagic and nutritional anæmias) are both secondary to a deficiency of one or other of the factors necessary for normal blood formation. No modern classification would be complete without revealing clearly that many of the anæmias are of the nature of nutritional deficiency diseases. In some anæmias the deficiency may be described as "direct," *i.e.*, due to an actual quantitative deficiency in the food; while in others it is indirect, *i.e.*, conditioned by a failure in absorption from the intestinal tract (sprue, idiopathic steatorrhœa, achlorhydric anæmia), or a

* Lecture delivered to the Harveian Society, London, November 19th, 1933.

failure in the manufacture of a principle essential for normal blood formation, *i.e.*, pernicious anæmia. Bearing these points in mind, it is possible to divide the anæmias into three main groups: (1) due to various deficiencies; (2) due to depression of or interference with blood regeneration (aplastic anæmia, idiopathic or secondary to toxic causes); (3) due to hæmolytic processes (congenital or acquired). Time does not permit me to deal adequately with the prevention and treatment of all three groups; accordingly I shall confine my observations to the most important group, *viz.*, the anæmias due to various deficiencies.

It is common knowledge that for the maintenance of health a diet must be taken which is adequate quantitatively and qualitatively. A deficiency of vitamins or minerals in the diet will lead to general systemic diseases which are frequently accompanied by some degree of anæmia. Likewise, anæmia may accompany certain secretory disorders of the endocrine glands. Such deficiencies, although they require adequate attention, are, however, relatively infrequent causes of anæmia, and they need not be discussed further in this paper. There are two factors which are essential for normal blood formation, and any cause which leads to a deficiency of either will invariably result in anæmia. Iron, which is essential for the formation of hæmoglobin, and the specific anti-anæmic factor which is required for stroma formation and the normal maturation of erythrocytes, must be available in optimal amounts. A brief study of how a deficiency of these factors can occur is therefore desirable.

A deficiency in iron may result from at least three causes: (1) a deficiency of iron in the diet; (2) defective absorption of iron from the gastro-intestinal tract; (3) increased demands by the organism for iron, due (*a*) to blood loss, (*b*) to pregnancy and lactation. The serious results of the prolonged ingestion of iron-poor diets are well recognised both in animals and human beings. When rats are fed on milk alone severe anæmia develops and growth and fertility are impaired. A similar anæmia may occur in infants fed for prolonged periods on the breast, and is even more frequently seen in those fed on the bottle. The diet of adults of the poorest classes often contains a large preponderance of carbohydrate and a relatively small proportion of red meat and green vegetables, which are the chief iron-containing foods. Investigations carried out in Aberdeen clearly indicate that the iron

content of such a diet is from one-third to one-half smaller than that contained in a well-balanced diet of the well-to-do classes. While iron-poor diets may maintain a normal blood level if no extra demands occur, they are quite unable to do so when any additional strain is put on the organism, as occurs in pregnancy, lactation, or when menorrhagia is present. Thus, of approximately 1,000 women of the child-bearing age nearly 50 per cent. were found to have some degree of anæmia; 15 per cent. of these women had a hæmoglobin level of 70 per cent. or lower. On the other hand, a reduced blood level was rarely found during the investigation of 750 children between the ages of five and fourteen years, and of adult males belonging to the same class as the women. While increased demands for iron, due to menorrhagia, pregnancy and lactation were found to be the primary causes of the anæmia, the long-continued ingestion of iron-poor diets played an important rôle by reducing the body's stores of the precursors of hæmoglobin with which to meet these contingencies, as was proved by the beneficial effects of iron therapy without any alteration of the dietary.

An adequate intake of iron may fail to maintain a normal blood level if absorption is defective. Absence of hydrochloric acid in the stomach (achlorhydria) undoubtedly plays an important rôle in this respect; for iron is better absorbed from the small intestine when the reaction of its contents is acid than when it is alkaline. In addition, the achlorhydric stomach has a rapid emptying rate, and it is probable that its contents are hurried through the small intestine, thus allowing less time for absorption to take place. Similar effects are produced by gastric operations, such as gastrectomy or gastro-enterostomy. Lastly, absorption is defective in chronic diarrhoeal diseases such as sprue and idiopathic steatorrhœa, or where there is a loss through fistulæ of the secretions of the liver or pancreas. The need for an additional iron intake when blood loss occurs is obvious. This applies especially to external hæmorrhage, where the loss of blood is absolute, and not so much to intravascular hæmolysis (hæmolytic anæmia), where 90 per cent. of the destroyed blood is retained in the body, where its constituents can be used again. Pregnancy and lactation produce effects which are comparable to those of external blood loss, since the foetus withdraws large amounts of iron from the maternal circulation, not only to form its blood, but also to stock

the foetal liver with the very rich stores of pigment complexes which are required for the manufacture of hæmoglobin during the first nine months of the infant's life.

A deficiency of the specific anti-anæmic factor leads to megaloblastic blood formation consequent on a failure of the normal maturation of red blood cells in the bone marrow. The specific anti-anæmic factor is found in liver and liver extract ; it is formed by the interaction of a thermolabile enzyme secreted by the stomach on some as yet unknown protein breakdown product of the food. A deficiency may arise in one of three ways: (1) a failure in secretion of the enzyme (intrinsic factor of Castle) ; (2) a deficient intake of protein foods (extrinsic factor) ; (3) a failure in absorption or a destruction of the specific anti-anæmic factor in the gastro-intestinal tract. Addisonian pernicious anæmia and the pernicious anæmia of pregnancy, and some cases of sprue, are due to a failure in gastric secretion. Insufficiency of extrinsic factor is of little importance in temperate climates, but appears to play an important part in the tropical macrocytic anæmia described by Wills. Examples of macrocytic blood diseases belonging to the third group are sprue, bothrioccephalus anæmia, and some cases of idiopathic steatorrhœa.

From what has been said, it is clear that the two great groups of anæmia due to deficiencies are entirely different from one another. As a rule it is as useless and extravagant to prescribe iron for the treatment of hyperchromic macrocytic anæmia as to order liver extract for hypochromic microcytic anæmia. If an accurate diagnosis is made, then treatment of the anæmias due to deficiencies becomes a simple matter.

Prevention.

(1) *Hypochromic Microcytic Anæmia*.—If the ætiological factors outlined above are appreciated, much can be done for the prevention of anæmia. Hypochromic anæmia is approximately ten times more common in women of the child-bearing age than in men or children. The deleterious effects of menstruation and pregnancy on the blood level can be markedly diminished by the ingestion in adequate amounts of iron-rich food. If for economic reasons a sufficiency of animal protein and green vegetables cannot be provided, a small maintenance dose of iron should be prescribed. By this means the anæmia of infancy, and the ill-health and susceptibility to disease which are its concomitants, can be prevented,

since the storage of iron in the foetal liver, on which the infant relies so much during its first year of life, depends on the maternal blood level during the last trimester of pregnancy. It stands to reason that the occurrence of menorrhagia, diarrhoea or chronic intestinal disease demands adequate medical attention, in addition to the symptomatic treatment of the anæmia.

Achlorhydria, causing defective absorption of iron, can frequently be treated with success. This entails: (1) dietary regulation, with the exclusion of irritating and indigestible articles, whether in the form of food or drink; (2) the removal of septic foci in the mouth, nose and throat; and (3) gastric lavage with a weak solution of hydrogen peroxide at night for several weeks. By such means many cases of simple achlorhydria can be cured.

(2) *Hyperchromic Macrocytic Anæmia*.—Insufficiency of extrinsic factor (protein foods) which could easily be corrected, plays a minor rôle in the pathogenesis of Addisonian pernicious anæmia. The essential defect, *viz.*, a failure in gastric secretion, can rarely be influenced by treatment, since it is permanently and completely established, in the great majority of cases, by the time diagnosis is made. A remarkable exception to this rule was, however, recently published by the writer. Nevertheless, there is much evidence to suggest that achylia gastrica is frequently the terminal stage of an antecedent achlorhydria consequent on long-continued chronic gastritis. Since the secretion of the intrinsic factor is lost at a later stage than hydrochloric acid, the principles already laid down regarding diet, focal sepsis and gastric lavage, if regularly employed in all cases of achlorhydria, should play a part in preventing or delaying the onset of some cases of pernicious anæmia.

Treatment.

Hypochromic Anæmia.—Since this type of anæmia is due to iron deficiency, treatment consists of iron replacement. While diet is of great importance in prevention, the quantity of iron which can be assimilated by this method is insufficient for therapeutic purposes. It is accordingly necessary to consider what preparation of iron should be employed, its manner of administration and dosage. If there is one lesson which recent investigations have clearly demonstrated, it is the need for massive doses of iron. My assistant, Dr. Fullerton, has undertaken a carefully controlled investigation into these points, and the conclusions, which he is

publishing separately, may be summarised as follows : Parenteral injection of iron salts cannot be recommended, since (a) this method is no more effective than oral therapy ; (b) pain at the site of injection is produced ; (c) the optimal dose is dangerously near the toxic dose. Accordingly, only the physician, and not the patient, benefits from parenteral iron therapy ! An investigation into the value of (1) iron and ammonium citrate (grains 90 daily), (2) Blaud's pills (grains 45 daily), (3) ferrous sulphate tablets (grains 9 daily), (4) ferrous chloride tablets (grains $7\frac{1}{2}$ daily), (5) ferrous carbonate tablets (grains 9 daily), clearly showed that in the doses stated all the preparations were equally satisfactory, since a rise in the hæmoglobin level of 1 per cent. or more daily was obtained. This is a very strict standard for passing a preparation as satisfactory. Dr. Fullerton's studies indicate, however, that there are great differences in the percentage utilisation of these preparations; thus 15 to 20 per cent. of the iron in the form of ferrous sulphate or chloride was utilised in the formation of hæmoglobin, compared to only 2.5 per cent. in the case of iron and ammonium citrate. This is in keeping with the modern belief that the efficacy of an iron preparation depends largely on the amount of ferrous ions liberated in the small intestine. Since the 180 milligrams of metallic iron in nine grains of ferrous sulphate was as effective as 1,200 milligrams of iron contained in 90 grains of iron and ammonium citrate, it follows that only a small proportion of the scale preparation must be turned into the ferrous state in the intestinal tract. As all the preparations mentioned above were found to be satisfactory in the dosage stated, the final choice of the therapeutic agent will depend largely on convenience and cost. For the past few years I have relied mainly on iron and ammonium citrate, 90 grains daily, with entirely satisfactory results. It is wise to start with 45 grains for a day or two, as colicky pains and diarrhœa are occasionally produced by bigger doses, especially in ambulatory patients. Recently, however, I have been using a tablet containing three grains of exsiccated ferrous sulphate, made for me by Glaxo laboratories ; this preparation is now on the market. Three tablets per day are effective, and patients prefer this to taking nine Blaud's pills. Three-quarters of a Blaud's pill consists of pill mass which tends to harden, and hence retard absorption in the intestine. A small bottle containing 100 tablets of ferrous sulphate will last a month, while a mixture containing iron and ammonium citrate

requires to be renewed every four days. In addition, blackening of the teeth and tongue results from the administration of fluid iron preparations, but does not occur when the metal is given in tablet form. Finally, from an economic standpoint, treatment with the ferrous sulphate tablets is the cheapest method available, costing about 1*d.* a day, compared to 3*d.* or 4*d.* with the other preparations mentioned above.

Hyperchromic Macrocytic Anæmia.—Where megaloblastic blood formation is due mainly to deficiency of extrinsic factor or defective absorption, as occurs in tropical macrocytic anæmia and in some cases of sprue and idiopathic steatorrhœa, a high protein diet combined with autolysed yeast products, such as marmite (which contains twenty times more extrinsic factor than beef protein), will be effective. In Addisonian pernicious anæmia, in which the essential defect lies in a failure of secretion of intrinsic factor, liver, liver extract or hog's stomach preparation must be employed. Since 1926 I have tested carefully most of the preparations issued by the leading firms in this country, the Continent, Australia and America, and have come to the conclusion that it makes little difference whether liver, liver extract or hog's stomach tissue is chosen, provided (1) potent preparations are employed, and (2) adequate doses are prescribed. The final choice depends on individual taste, convenience and cost.

There is one aspect, however, which I wish particularly to stress, namely, the importance of envisaging the treatment of pernicious anæmia on a quantitative basis, so that adequate supplies of potent material should be received by the patient to meet his optimal daily demands throughout life. This entails not merely a maintenance of a blood count at 5 million red cells and 100 per cent. hæmoglobin, but the storage of adequate amounts of the specific anti-anæmic factor in the body to meet eventualities, *e.g.*, infections, gastro-intestinal upsets, etc. The amount of specific anti-anæmic factor required for this purpose varies in different cases; thus a patient in his first relapse who has had the disease only a short time may respond adequately to 100 or 200 grams of liver, or the extract made therefrom; while an elderly patient who has had pernicious anæmia for several years, especially if his state is complicated by arterio-sclerosis or chronic sepsis, may require from 500 to 1,000 grams of liver daily, or the equivalent of extract. Exactly the same considerations are applicable to

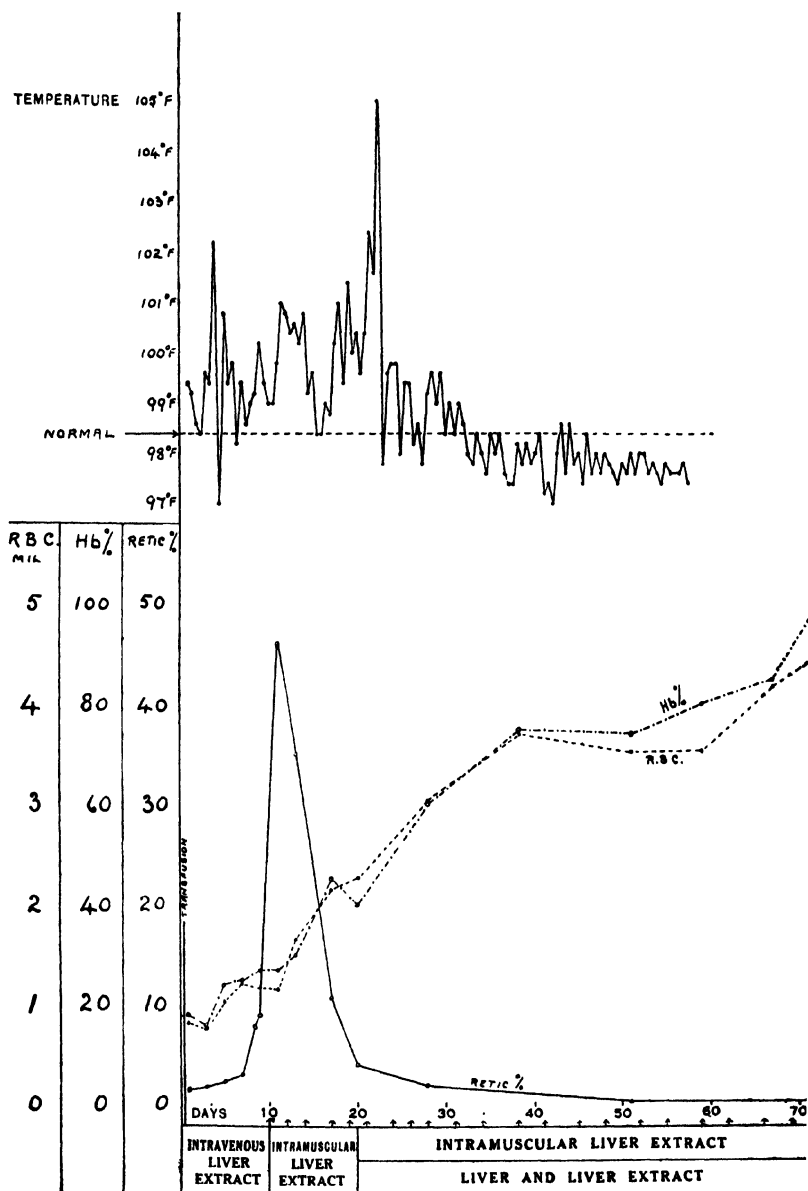
parenteral liver extract therapy, in which the amount of active principle injected will vary in different cases from 10 to 100 grams of liver daily. It is a calamity that the view should be so widely held that half a pound of liver on several days a week is *the* therapeutic dose. Worse still is the belief that the extract of half a pound of liver can be substituted for half a pound of whole liver, since it is certain that extraction removes only about 60 per cent. of the active principle. To suggest that any weighted quantity of liver or liver extract is the universal therapeutic dose is as ridiculous as to state that one grain of thyroid extract should be given to all cases of myxœdema, or that thirty units of insulin should always be prescribed in diabetes. Just as the dose of thyroxin is regulated by a study of the patient's metabolism and the dose of insulin by the results of blood and urine sugar analysis, so the dose of specific anti-anæmic factor, whether in the form of liver, liver extract or gastric-tissue products, must be regulated by repeated estimations of the blood level. The need for regular blood examinations is brought home to me frequently in the Aberdeen Blood Clinic, where indisputable evidence is at our disposal which shows that not only do oral and parenteral preparations of liver made by different commercial firms vary in potency, but different batches issued at intervals by the same firm may show wide differences in activity. A failure to recognise the above principle is responsible for the tragic increase in the number of cases of pernicious anæmia suffering from subacute combined degeneration of the cord. Another aspect of treatment also requires the greatest emphasis. It is widely believed that while large amounts of specific anti-anæmic factor should be given during the stage of relapse when the blood count is low, the dosage can be reduced in two or three weeks when the patient has improved. From prolonged observations I am satisfied that the reverse is the case: at least twice as much specific anti-anæmic factor must be given daily to raise the blood count from $3\frac{1}{2}$ to 5 millions as to raise the blood level from 1 to 3 millions. In consequence, many cases of pernicious anæmia are carrying on their daily vocations with a red-cell count around 4 millions and hæmoglobin level of 85 per cent., looking and feeling well. It is from their ranks that the cases of subacute combined degeneration come. Only blood examinations carried out at regular intervals can reveal this dangerous state, since a 15 to 20 per cent. reduction in the blood level cannot be recognised

by clinical examination alone. To raise the blood count from 4 to 5 million red cells and to stock the body stores adequately with specific anti-anæmic factor may be very difficult by oral therapy alone, especially in elderly patients who have had the disease for some years. We have cases of pernicious anæmia reporting to the Aberdeen Blood Clinic in which half a pound of liver on six days a week, or the ingestion of extract from 250 to 500 grams of liver, or two heaped tablespoonfuls of Pepsac daily, failed to attain or maintain the optimal level of 5 million red blood cells and 100 per cent. hæmoglobin. Under these circumstances parenteral therapy, which is thirty times as effective as oral therapy, must be instituted, when a satisfactory result can be expected.

In conclusion, it may be said that if oral treatment is the method chosen, whole liver (containing as it does a rich store of iron and copper, pigment complexes and aminoacids of high biological value) should be the main basis of the regimen. It can be lightly cooked in a large variety of ways. In the majority of cases half a pound of liver alternating daily with the extract from 250 grams of liver, or two tablespoonfuls of hog's stomach tissue, *e.g.*, Pepsac, will be sufficient; but this dose must be increased or replaced partly or in whole by parenteral treatment if repeated blood counts show that it is insufficient to maintain a completely normal blood level. With regard to cost, whole liver and hog's stomach preparations made in this country are considerably cheaper than liver extract. Gastric tissue products, however, have minor disadvantages. They are insoluble and thermolabile, and hence must not be heated. In addition, they have an unpleasant taste and odour, which, however, can be materially reduced by exposing the next day's dose to the air for twelve hours.

Parenteral Liver Therapy.

The injection treatment of pernicious anæmia has now definitely passed out of the experimental stage, and the possibility of its use must be carefully considered in every case of pernicious anæmia. There are three clear indications for its employment: (1) The acute stage of relapse when the patient is so seriously ill as to be in danger of his life: the speed of blood regeneration which results from parenteral liver therapy is of paramount importance. (2) In resistant cases it may be of value in raising the blood level to normal. (3) For the replacement, partly or in whole, of treatment



A case of pernicious anæmia with double pneumonia and gangrene of the back successfully treated by parenteral liver extract therapy.

with liver or liver extract by mouth during the stage of remission ; this aspect is of special importance, since treatment must continue for life. While the effect of sepsis in retarding or stopping the therapeutic action of liver preparations given by mouth is well known, it is not generally recognised that parenteral therapy can act effectively in the presence of even the most intense toxæmia. Thus, a patient of mine who had a blood count of 600,000 erythrocytes, severe heart failure with widespread œdema, double pneumonia and gangrene of the back, responded to intravenous therapy with a 46 per cent. reticulocyte crisis, and a rapid rise in the blood level. Liver extract may be given intravenously or intramuscularly. While the latter route will naturally appeal to the general practitioner, and will usually be his method of choice, it is desirable to state the advantages and disadvantages of both methods.

Intravenous Therapy.

If intravenous therapy is to be given, only specially purified preparations, free from protein and histamine and similar shock-producing substances, must be employed. The advantages of the intravenous route are : (1) There is less pain to the patient than from intramuscular injection. (2) The physician is sure that all the active principle has entered the blood stream : in patients critically ill, with a failing circulation and much subcutaneous œdema, intramuscular injections may be of little value, owing to failure of absorption, and a fatal result may ensue before adequate amounts of the active principle reach the bone marrow. (3) There is great rapidity of action : the reticulocytes begin to increase within twenty-four to forty-eight hours, reaching a peak within a week. (4) The excellence of the hæmatological response, which is generally maximal.

The disadvantages are : (1) The inherent difficulty common to all forms of intravenous therapy. (2) The danger of sepsis unless full precautions regarding sterility are taken. (3) The dangers of severe reaction. These can be largely guarded against by selection of specially prepared products, and by giving the injection very slowly. Symptoms of dyspnœa and falling blood-pressure can usually be rapidly relieved by the injection of adrenaline.

The dosage employed by different workers varies considerably, depending on the potency of the preparations employed ; but the majority inject the active material obtained from 50 to 100

grams of liver on three or four consecutive days, by which time a satisfactory rise in the number of circulating reticulocytes should have occurred, and continue thereafter with an injection once weekly. The maintenance dose of any one preparation varies in different patients, but many keep in normal health when the active principle from 20 to 100 grams of liver is injected once every two weeks. Some cases require a weekly injection, while others maintain a normal blood level over long periods on a monthly injection. Only by repeated blood examinations is it possible to specify the dosage and the time intervals between injections. It is a wise principle in all forms of substitution therapy to use small doses at more frequent intervals, rather than large doses at long intervals.

Intramuscular Injection.

The advantages of this method to the general practitioner are obvious. Any pain at the site of injection may be largely relieved by massage for a few minutes in order to distribute the fluid through the tissues. General reactions very rarely occur, and the speed of blood regeneration is very rapid. The slightly slower response obtained, compared to that produced by intravenous injection, is usually of little importance, except in cases which are desperately ill. Practitioners must remember that while preparations for intravenous use may be given intramuscularly, the reverse must never be done. Intramuscular preparations are considerably cheaper than intravenous ones, since the latter must be subjected to an extensive process of purification. Effective preparations for intramuscular injection can be made very simply in any laboratory, since the active principle contained in liver extract (fraction G, Cohn) has merely to be dissolved in distilled water, suitably buffered and sterilised thereafter. Since this simple method does not concentrate the material, few of the preparations for intramuscular use contain the active principle from more than 5 to 10 grams of liver per c.c. The extract from 20 to 40 grams of liver daily is required for treatment during the acute stage. For the maintenance of a satisfactory blood level, the active principle from 20 to 50 grams of liver injected once a week will generally be found to be sufficient. Many case reports have now been published which showed a failure to respond to the oral administration of extract from 200 to 800 grams of liver daily, but which reacted satisfactorily to the injection of the active principle derived from 20 to 100 grams.

There are two important reasons which are inducing more and more of those who have made a special study of the treatment of pernicious anæmia to suggest the replacement of oral therapy, wholly or partly, by parenteral: (1) Eight years have passed since liver treatment became established, and large numbers of patients are alive to-day who previous to 1926 would have died. Many of these patients, through ignorance, laziness or inability, have been taking insufficient quantities of potent material, with the result that subacute combined degeneration of the cord, once a relatively rare disease, is now of common occurrence. Since treatment of subacute combined degeneration, except in early cases, is unsatisfactory, prevention becomes of paramount importance, and the continuous maintenance of a normal blood level offers the best hope in this direction. The need for adequate therapy based on repeated blood examinations is clearly evidenced by the statistics of cases of pernicious anæmia treated at the Royal Hospital, Wolverhampton. Of 52 cases treated within the last five years, 19 attended regularly after discharge for blood examination and advice, all of whom are alive and well. Of the 33 patients who failed to turn up regularly for examination, only nine are in good health, and 15 are dead. These figures clearly indicate the essential need for treatment based on regular blood examination, and require no further comment by the writer. The injection of liver extract at regular intervals, either as the sole method of treatment or combined with the ingestion of moderate amounts of whole liver, is probably the safest way to attain this object. (2) From an economic point of view parenteral therapy is probably twenty to thirty times as effective as oral treatment, and a weekly injection of the active principle from 50 to 100 grams of liver is infinitely the cheapest method of treatment. There appears to be little reason why intelligent patients should not be taught to inject themselves in the same way that diabetics do.

CHAPTER LIII.

CHRONIC HOARSENESS

BY

HERBERT TILLEY, B.S., F.R.C.S.

Consulting Surgeon (Diseases of Ear, Nose and Throat), University
College Hospital, London.

SOME alteration in the voice of the individual may occur at any period of life from the cradle to the grave. In one case the symptom may be little more than a temporary inconvenience, as in simple chronic laryngitis; in another it will often be an early sign of the gravest significance, *e.g.*, primary cancer of a vocal cord, tuberculosis of the larynx, or paralysis of a vocal cord due to involvement of its recurrent laryngeal nerve by an aortic aneurysm, mediastinal growth, or to some central lesion involving the origin or course of the nerves which supply the intrinsic musculature of the larynx.

In a considerable experience of the diseases of the upper and lower air passages, it has often seemed to me that the clinical significance of chronic hoarseness is more frequently overlooked by members of our profession than any other subjective laryngeal symptom. This may possibly be explained by the fact that many of such patients had otherwise enjoyed good health, and did not complain of any local discomfort or "pain in the throat." For the sake of convenience it may be well to glance briefly at the most frequent causes of prolonged loss of voice as they may occur while life unfolds itself between infancy and "second childhood." The subject is a large one, and as the space at my disposal is so limited, it will not be possible to do more than hint at questions of diagnosis, and still less to discuss the treatment of the many conditions which may cause hoarseness.

Congenital Syphilis.

The hoarse cry of a newborn child, or the supervention of that symptom in the course of a few weeks after birth, should arouse suspicion of inherited syphilis. This would be strengthened by

the presence of a wizened look and earthy-brown coloration around the eyes, nose and mouth, and such symptoms as "snuffles," nasal obstruction and muco-purulent discharge from the nostrils. Furthermore, there may be eruptions on the buttocks or on the soles of the feet, and possibly "mucous tubercles" around the anus.

Treatment.—Mercury in some shape or form will be indicated both for mother and infant, and for the latter, inunction is a favourite method. But of almost equal importance, and possibly of greater difficulty, will be the nutrition of an unusually ill-conditioned infant of low resisting powers. The nasal obstruction will frequently prevent nursing at the breast, but sometimes it may be overcome by injecting into each nasal cavity about five minutes before feeding time, two or three drops of a 1 per cent. solution of cocaine in olive oil. This will bring about temporary contraction of the nasal mucosa and permit of normal breathing. Emphasis should be laid on the need for keeping such infants warm because of the poor fight they make against even the mildest forms of bronchial or pulmonary infections.

Papillomata of the Larynx.

These growths are more frequently seen in children than in adults, and they should be suspected when chronic hoarseness and cough are predominant symptoms in an otherwise healthy child. Stridor may develop if and when the papillomata increase in size or number and thus encroach on the glottic air-way, and this symptom may suddenly become urgent with the onset of a "cold in the head." In such circumstances a low tracheotomy would be an immediate necessity.

This combination of hoarseness and stridor in young children has sometimes led to a "snap" diagnosis of laryngeal diphtheria. I remember a case in point where a little boy, aged four years, was hurried into a hospital with those symptoms and some cyanosis. He was admitted into the diphtheria ward, tracheotomised, and anti-diphtheritic serum injected. The following day the patient appeared to be quite well, and was playing with his toys, so it seemed reasonable, in the absence of any general symptoms of toxæmia, to question the accuracy of the diagnosis. Inspection of the larynx showed multiple papillomata, which on subsequent occasions I removed, and finally they ceased to recur.

Treatment.—These growths should be removed with forceps and by direct endoscopy; it is the only method worthy of serious consideration. However thoroughly the operation be performed, they will probably recur and require further removal, and the parents should be warned of this possibility; but they may be equally assured that eventually the growths will not return. Probably, like warty growths in other regions, laryngeal papillomata are locally infective, and only disappear when a sufficient degree of immunisation has been established.

“Breaking of the Voice” at Puberty.

At this age the passage from the natural voice of a boy into that of the young adult is often accompanied by congestive changes in the larynx which cause a certain amount of gruffness almost amounting to “hoarseness.” Like so many other human affections, Shakespeare seems to have recognised this one, as expressed in the lines :

“ And speak between the change of man and boy
With a reed voice . . . ”

—*Merchant of Venice*, III, 4.

It is a physiological change and often accompanied by turgescence of the nasal mucous membranes. To interfere with these by galvanic cauterisation of the turbinals, or, what is worse, to remove harmless tonsils, are thoughtless and unjustifiable procedures. The cause and nature of the symptoms should be explained to anxious parents, and an assurance given that in the course of a month or two the voice of their boy will settle down into that of the young adult.

Functional Aphonia.

In this condition the sound of the voice is lost, and the patient only whispers, and often does so quite clearly. The condition is most frequently met with in the female sex from puberty to the menopause. It is caused by paresis of the adductor muscles of the cords. If the larynx be examined in the mirror it will be noticed that on attempted phonation the cords fail to approximate closely, so that an elliptical glottic aperture results. It is easy to be put off one's guard, because sometimes the symptom occurs in apparently healthy young women, while in other instances the aphonia will follow a slight attack of tonsillitis or laryngitis, prolonged illness, or as an immediate sequence of a sudden physical or mental shock.

During the Great War functional aphonia was very common in soldiers who had been exposed to severe "shelling" or who had been temporarily buried as a result of a "mine" explosion; otherwise it is uncommon to meet the symptom in the male sex. The diagnosis is usually easy, because if the patient be asked to cough, the sound of the voice is produced in normal fashion, *i.e.*, the adductors for the moment come into close apposition.

Treatment.—Functional aphonia can nearly always be cured by "suggestion," or by some unexpected mental or physical disturbance. In my experience the following method of "suggestion" rarely fails to immediately bring back the normal voice: Having sent the mother or other sympathetic friend out of the room, the patient is told that the little muscles in the voice-box are weak, and that the paint applied to them will restore the voice at once. The end of a laryngeal probe is then tipped with wool soaked in some simple pigment and, guided by the mirror, is passed through the glottis and held there for a moment. On its withdrawal the patient is asked to swallow some water, and is then told that as the voice has now returned, she is to count aloud from one to six. As a rule this can be done, and not infrequently her joy (or disappointment) is demonstrated by a few tears, and all is well. Let us not be so unchivalrous as to remember Hamlet's observation: "Frailty, thy name is woman."

Should this method not succeed, the larynx is again examined, and one states with assurance that some weakness still remains, but an "electric spark" will cure it. The application of a strong Faradic current between the intra-laryngeal electrode and the external terminal over the larynx will very rarely fail to produce the desired effect. These procedures illustrate the value of some slight physical shock; but a sudden mental disturbance is often equally efficient, *e.g.*, during the Great War a soldier of a Scottish regiment was on leave and suffering from functional aphonia, but immediately regained his voice when, at a ticket office, he thought his change was short by twopence!

Vascular Fibroma of the Vocal Cord.

This small purple-red, sessile or pedunculated and benign tumour grows from near the edge of a vocal cord, and usually at the junction of its anterior and middle thirds. It is more common in males from thirty to fifty years of age. The growth may vary in size

from a hemp seed to a large pea, and causes various degrees of interference with the normal voice. When effectively removed by forceps it does not recur if the patient can be induced to only whisper for at least a fortnight after the operation.

“Teachers’ Nodes,” or “Singers’ Nodules.”

These are very small, discrete, whitish projections from the edges of the cords at the junction of their anterior and middle thirds. They are probably caused by the friction of the cords at these nodal points, when the voice is used for long periods of time, and at too high a pitch. They are far more common in the female than in the male teachers of our Council Schools. Incidentally, they involve considerable cost to the ratepayers because of the long period of rest which treatment involves and the necessity for the provision of a paid substitute. Teachers and singers suffering from such nodules find that the voice quickly tires, and its further use induces pain in the larynx and complete hoarseness. I have seen a few cases in quite young choir boys.

“For my voice I have lost it with holloing and singing of anthems.”

—*Henry IV*, Part 2, I, 2.

Treatment.—The only satisfactory method is prolonged vocal rest, followed by a course of training given by an expert in voice production. In very advanced cases, *i.e.*, where the nodules are larger than usual, the application to them of a very fine galvanocautery point may hasten the cure.

Chronic Laryngitis.

This is an affection of both vocal cords, and is most frequently seen in adults. In a typical case the laryngeal mirror will show that the pearly-white upper surfaces of the vocal cords have been transformed into dull reddish bands with rounded borders, and in parts covered with dark inspissated mucus. The interarytenoid fold and ventricular bands often share in the general congestion of the laryngeal mucosa. Chronic laryngitis is almost always the result of one or more unhealthy conditions outside the larynx itself. Perhaps it would be true to say that the large majority of cases results from the spread of catarrhal conditions affecting the nasal and nasopharyngeal cavities, and the more so when septic foci are present in those regions, *e.g.*, in the nasal accessory sinuses or in the remains of old adenoids. Similar factors may exist in the

teeth, tonsils, and unclean dentures, especially when the latter are not removed at night, and thus provide a perfect incubating chamber for all kinds of septic organisms. Such influences will be exaggerated if the patient has to work in dusty or badly ventilated surroundings, and particularly so if his occupation entails excessive use of the voice, or if there be undue indulgence in tobacco and alcohol.

With regard to the latter, patients almost always minimise their addiction. The Psalmist was right when he said: "All men are liars," and had he lived in these latter days he would have included women in his wrathful generalisation. Finally, whatever may be the local factors, their action will be promoted by any constitutional disease, *e.g.*, syphilis, gout, rheumatism, diabetes, albuminuria, arteriosclerosis, etc.

Treatment.—Independently of the underlying cause of the chronic laryngitis, it must be insisted, as a first principle of treatment, that functional rest be given to the vocal cords, for without this the success of any other measures will be severely handicapped. With regard to local treatment of the larynx, solutions of nitrate of silver, from 5 to 20 gr. to 1 oz., are the best, but usually they can only be applied by an expert laryngologist.

Syphilis of the Larynx.

This is usually seen as a tertiary manifestation, and takes such forms as gummatous infiltration, ulceration, perichondritis, necrosis of bone or of cartilage, scars and adhesions. The infiltrating gumma is usually a smooth, rounded, irregular and deep red swelling, and it may be more or less localised to any part of the larynx. If the structures around the glottis be involved some degree of stridor may result. In a typical gummatous ulcer its base is often covered with a greyish-yellow slough surrounded by a well-defined hyperæmic border (Fig. 1). Hoarseness will result if the vocal cords be infiltrated or ulcerated, or if their movement be hampered by neighbouring lesions, *e.g.*, by involvement of the corresponding arytenoid articulation.

Treatment.—This will be on general lines of treatment for tertiary syphilis. It must be remembered that when extensive infiltration or ulceration heals, some cicatrization will probably result and render the hoarseness more or less permanent. Of this possibility the patient should be warned.

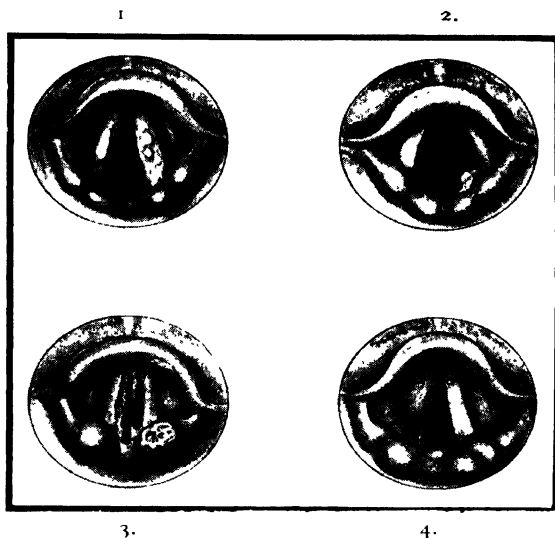


FIG 1.—Syphilis of larynx. 1. Small greyish white, oval or round infiltrations on vocal cords. 2. Destruction of left vocal cord, remains of processus vocalis still visible. 3. Typical serrated ulceration of edges of vocal cords and characteristic punched-out ulcer of left arytenoid. 4. Gumma of right ventricular band hiding the corresponding cord.

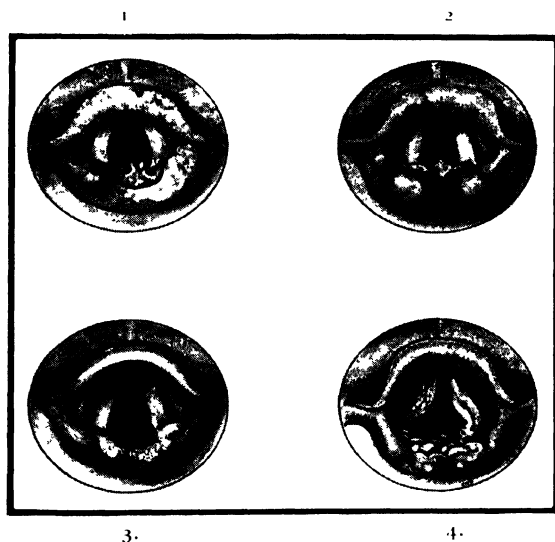


FIG 2.—Tuberculosis of larynx. 1. Superficial ulceration of epiglottis and left arytenoid region. Note the prominent granulation in the interarytenoid region. 2. Swelling of arytenoids and ulceration region of interarytenoid region. 3. Superficial ulceration of left vocal process and arytenoid region. 4. Extensive ulceration of cords, interarytenoid and arytenoid regions.

"CHRONIC HOARSENESS," by Herbert Tilley, B.S., F.R.C.S.

Tuberculous Laryngitis.

This may be regarded as always secondary to a primary lesion in the lung. I have yet to see a case of primary tuberculosis of the larynx. The lesions may take the form of infiltration, ulceration, necrosis of cartilage and, in rare instances, of a localised "tuberculoma" simulating a new growth.

Between early and advanced manifestations of tuberculous laryngitis the gradations are so numerous that it would be impossible to describe them all on this occasion, and therefore it is only necessary to mention a few common varieties of early infection: (*a*) a granular congestion of one vocal chord (Fig. 2) is especially suggestive of tubercle in a young and delicate patient who, in addition to hoarseness, complains of a "slight cough," want of energy, and whose pulse-rate, even when resting, is unduly rapid; (*b*) an unilateral, superficial, irregular ulceration on the anterior aspect of the interarytenoid fold; (*c*) a small, pale, shallow ulcer on the vocal process, or on the posterior third of the vocal cord; (*d*) the latter, with slight pseudo-œdema of the corresponding arytenoid; (*e*) in more advanced cases, a pale, superficial and more generalised "mouse-nibbled" ulceration involving the ventricular bands and one or both cords (Fig. 2).

In still more extensive lesions the ulceration may spread to the epiglottis, while the glottic regions of the larynx become so involved in a widespread, pale, superficial and irregular ulceration that it may be impossible to define the outlines of normal structures (Fig. 2).

Prognosis.—Laryngeal tuberculosis must always be regarded as a serious complication of the pulmonary lesion. Speaking generally, it is probably true to say that the extent and progress of the disease as seen in the laryngeal mirror is a fair reflex of what is going on in one or both of the lungs. On the other hand, if general treatment brings about a definite improvement in the laryngeal lesions, it may be assumed that the pulmonary mischief is also on the mend.

Treatment.—In their earlier stages both the pulmonary and laryngeal lesions may often be cured or rendered quiescent for long periods by sanatorium treatment or by the various forms of "light" treatment. Whatever be the general method adopted, vocal rest will be imperative in so far as the larynx is concerned, and it has often proved to be successful without the employment

of any topical applications. If, however, the laryngeal trouble lags unduly while the lung conditions improve, galvano-puncture of an ulcer or infiltration will frequently stimulate a healing fibrosis.

Cancer of the Larynx.

Squamous-celled epithelioma is the most frequent form of malignant disease of the larynx. It is ten times more frequent in men than in women, and is met with about twice as often between the ages of fifty and sixty than in the preceding ten years of life. When the lesion affects a vocal cord or the structures in its immediate neighbourhood, it is spoken of as an "intrinsic" epithelioma, and the earliest symptom of its presence is hoarseness or some alteration in the normal voice of the patient. "Extrinsic" growths are those which commence on the epiglottis and other laryngeal structures beyond the regions in close proximity to the glottis, and as they do not affect the voice in the earlier stages of their growth, no further reference to them will be necessary.

As seen in the laryngeal mirror, there are at least three more or less common types of intrinsic epithelioma of a vocal cord : (1) a dull, greyish-white, sessile, warty-like nodule on the middle third of the upper surface and edge of the cord ; (2) an irregular, congested, granular ulceration dotted here and there with small whitish-grey spots due to maceration of epithelial cells ; (3) a much less common type than either of the former is seen when the upper surface and edge of the cord presents a white shredded appearance, which has been compared with the pale stalks of freshly-mown grass. (Figs. 1, 2, 3, 4.)

On occasion all these appearances may be seen in varying proportions in an individual lesion. In the earlier stages of an epithelioma the affected cord will often be seen to adduct freely on phonation and abduct with deep inspiration, but later on when the sub-epithelial structures have been penetrated and the muscular elements of the cord invaded, it will be noted that such normal movements are hampered, and eventually the cord may become immobile.

The inference to be drawn from these observations is that when an adult suffers from chronic hoarseness, and a growth which to the experienced eye looks suspiciously like an epithelioma is seen on one of the vocal cords, the fact that the latter moves freely on

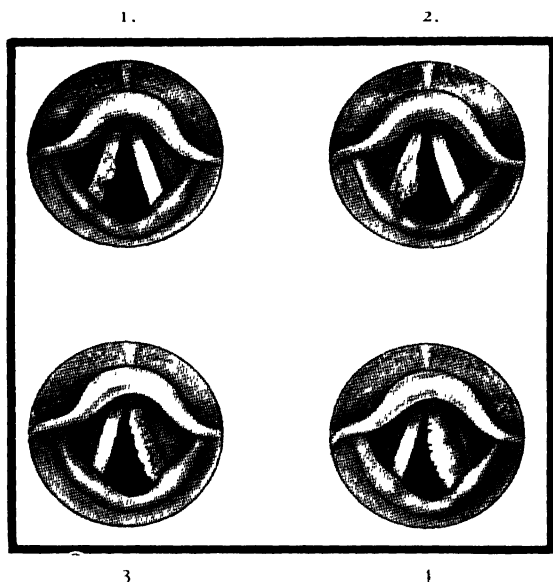


FIG. 3 - These figures illustrate some of the commoner forms of intrinsic epithelioma of the larynx. 1. Small nodule on right vocal cord. 2. Larger nodule surrounded by slight zone of congestion. 3. Infiltration of left vocal cord presenting a granular appearance. 4. Infiltration of left vocal cord, in which the growth somewhat resembles the pale stems of new-mown grass. (*Drawn by S Sewell.*)

"CHRONIC HOARSENESS," by Herbert Tilley, B.S., F.R.C.S.

attempted phonation would be regarded as a very favourable feature because it implies that the disease is in its earlier stages. In such circumstances a hopeful prognosis of treatment may be given. On the other hand, if the lesion be seen on an immobile cord, it may be inferred that its deeper structures have been invaded and to an unknown depth. This may call for a more guarded opinion, in view of the possible and severer measures which may have to be adopted for complete removal of the growth.

I have laid some stress on this question of the mobility of a vocal cord when a new growth is seen upon it, because it was formerly taught that the outstanding sign of malignancy was impaired action of the affected cord. This often led to unnecessary delay, and the case was "watched" till the pathognomonic sign of defective movement declared itself. Nowadays, if the clinical appearances of the new growth on a freely moving cord are strongly suggestive of malignancy, we do not delay treatment for the relatively later sign of hampered action to make its appearance.

The lesson to be drawn from these considerations on intrinsic cancer of the larynx is that "when an adult suffers from chronic hoarseness of more than a month's duration he should be urged to submit to a skilled examination of the larynx."

In no region of the body is the treatment of *early* epitheliomatous cancer more hopeful than when this disease affects a vocal cord, and while this holds true, even when the muscular elements have been invaded and the cord rendered immobile, it will be obvious that the earlier the diagnosis has been made and treatment accorded the better will be the chances of freedom from recurrence for periods varying from 5 to 20 years. To delay interference until the advent of pain or stridor will be to rob the patient of his best chances of such freedom, and even if this end be secured it will often involve the risk of extensive and mutilating operations.

Treatment.—At the present moment the most favoured operation is laryngo-fissure, *e.g.*, splitting the larynx in the middle line, full exposure of the growth, and its radical removal. On the other hand, there are those who argue that a much better voice can be secured if a "window resection" of the thyroid cartilage be made, and a palisade of radium needles be inserted against the perichondrium on the outer aspect of the affected vocal cord.

Paralysis of the Vocal Cords.

Not infrequently chronic hoarseness is caused by paralysis of one or both vocal cords. It will be remembered that with the exception of the cricothyroids and the depressors of the epiglottis, all the other intrinsic muscles are supplied by the recurrent laryngeal branches of the vagus. On the left side the recurrent nerve has a long course before it leaves the main trunk of the vagus to turn beneath the arch of the aorta and course upwards between the trachea and œsophagus to its termination in the corresponding laryngeal muscles. Hence it runs the gauntlet of more evil possibilities than the shorter right recurrent nerve, and consequently paralysis of the left vocal cord is much more frequent than that of the right. Furthermore, in seeking for the cause of a paralysed cord, we must visualise the possibility of a lesion anywhere between the nuclear origin of the corresponding vagus and the termination of its recurrent laryngeal branch.

Many years ago, by his clinical observations, and later on by experiment in conjunction with Victor Horsley, Felix Semon pointed out "that in all progressive lesions of the centres and trunks of the motor laryngeal nerves, the abductors of the vocal cords succumbed much earlier than the adductors." This is known as Semon's Law, and it still holds good.

As a consequence, in the early stages of a central or peripheral lesion involving a recurrent laryngeal nerve, the corresponding paralysed cord will be motionless near the midline because of the unopposed action of the adductor muscles, and only later on, when these become affected, will the cord pass into the cadaveric position, in which the glottis is about half as wide as that seen in quiet respiration. Time will not allow me to review the various lesions which may involve the central origin of the recurrent laryngeal nerves, but I have seen cordal paralysis in progressive bulbar paralysis, basal pachymeningitis in the region of the jugular foramen, syringomyelia, disseminated cerebro-spinal sclerosis, and in tabes. Peripheral causes are more frequent than central because of the long course of the left recurrent nerve already referred to.

In practice, if in a case of chronic hoarseness it is seen that one vocal cord is paralysed and that the patient has no symptoms of a central nerve lesion, the chest should be examined for physical signs of aneurysm, and if this fails the thorax must be "screened," because a small aneurysm in the concavity of the aortic arch may

only be detected by this method. Furthermore, radiography may disclose a mediastinal tumour or a thickening of the pleura in the right apex. Should the patient, in addition to the hoarseness, complain of difficulty in swallowing, it is possible that the primary lesion is a carcinoma of the gullet involving the recurrent nerve, or pressure on it by infected mediastinal glands.

On one occasion, and from this cause, I saw a patient in whom both recurrent nerves were involved, so that not only was the voice affected, but stridor was a prominent symptom because the abductor muscles on each side were paralysed. The diagnosis was not easy, because there were no definite physical signs of intrathoracic disease, and the patient had no difficulty in swallowing. Yet at the subsequent post-mortem there was a large non-obstructive, malignant œsophageal ulceration with numerous infected mediastinal glands which had involved both of the recurrent laryngeal nerves.

In conclusion, I venture to hope that this brief outline of some of the commoner causes of chronic hoarseness will illustrate the clinical significance of a symptom which is too often regarded as of little importance, and thereby involves the loss of valuable time, which for the patient may mean " Too late, too late ! "

CHAPTER LIV.

CLINICAL FEATURES AND TREATMENT OF ELECTRICAL INJURIES

BY

HILARY LONG, B.Sc., M.B., B.S.

Diploma of University of Vienna ; Travelling Student of the
University of London, 1932-33.

THE symptoms and signs which accompany and follow electrocution of the human body are numerous and diverse, depending on several factors, the most important of which are: (1) the amount of electrical energy passing through the organism, *i.e.*, voltage multiplied by amperage of the current; (2) the type of the current; alternating currents of a low periodicity causing the more severe disturbances; (3) the electrical resistance of the body at the moment of contact.

Nevertheless, it is possible to classify the accidents more commonly seen into three main groups: (a) Patients in whom the general functional effect predominates; (b) patients who exhibit more or less localised external lesions; (c) patients whose signs and symptoms are due to invisible or internal effects; (d) patients who show any combination of the previous three.

It is my purpose to give a short account of the more important clinical features and treatment of the first three groups, illustrating them with cases which I am able to publish by the courtesy of my teacher, Professor Jellinek, of Vienna.

Group (a).

The shock accompanying every electrical accident is due to the functional effect of electricity on the body, and is manifested in varying degrees of intensity, from a deep momentary period of shock or fright to a state of deep unconsciousness. Should this condition persist for several minutes the patient passes into the dangerous state

known as suspended animation, lying as one dead, pulseless, with no obvious signs of respiration and no audible heart sounds, and a fatal termination is possible. No clear explanation of these clinical phenomena has so far been advanced, but they are believed to be due to the sudden and paralysing effect of the accident on the vital centres in the medulla, leading to a temporary or permanent derangement of the vital functions. However severe the shock, suitable treatment must be immediately undertaken, for the prognosis of the case is largely dependent on the rapidity and efficiency with which this is applied.

Treatment.

Artificial respiration—preferably by Silvester's method—must be begun on the scene of the accident, and continued for at least an hour, even in the apparent absence of success. If unconsciousness persists and conditions permit, a lumbar puncture should be performed at the end of the first twenty minutes to relieve the rise of cerebro-spinal pressure, which is believed to be a feature of the more severe cases. The cerebro-spinal fluid is allowed to escape until the pressure is again normal. Immediate return to consciousness may follow, or it may be necessary to resume artificial respiration.

Should the patient relapse into unconsciousness within the next twenty-four to thirty-six hours, lumbar puncture should be repeated.

It sometimes happens that the patient responds to prolonged artificial respiration without immediate lumbar puncture, but develops signs of cerebral irritation at any period within twenty-four hours. This must be treated by immediate lumbar puncture, or the patient may lapse into unconsciousness and die. Application of counter irritants over the heart and anus is beneficial as a general stimulant. When the dangerous stage is passed the patient must be put to bed in a darkened room, given an aperient, and kept on a light diet for several days.

Case 1.—This case is quoted to illustrate the severe functional effect of electricity. The patient was electrified while at work, the current passing through both hands and out through his feet. He was rendered unconscious, and brought to hospital. On arrival, the patient, who was a strong, healthy man, aged forty, lay quietly on the bed with a dazed expression on his face, as if he did not know where he was. — The pulse was increased in rate and tension. When

he was ordered in a loud voice to move any part of his body, he did this correctly after a prolonged latent period, but when asked to tell us his name, he either could not remember it or was unable to enunciate it, and did not speak at all in spite of repeated interrogation.

The reflexes were all slightly exaggerated, and the patient had an Argyll Robertson pupil. There were no local lesions: merely a faint marking where the current left the body on the soles of the feet.

This whole clinical picture, as we have seen, we believe to be due to a cerebral œdema, which is also present in the spinal cord. This patient was kept under the closest observation for the next few hours because he was the type of case who might after several hours develop Jacksonian epilepsy, lapse into coma and die. This can be avoided if immediate lumbar puncture is performed at the onset of the symptoms. The patient, however, slowly recovered from the functional disturbance without the necessity for any further interference; by the next day was very much better, and was able at the end of a few days to leave hospital.

These cases of functional disturbance present a clinical picture which when once seen are readily recognised.

Group (b).

The external lesions vary according to the area of contact and the amount of electrical energy which enters the surface of the body, the smallest and commonest being the electric current mark which is found on the skin at the points of entry and exit of the current. These marks are usually seen on the hands or feet, and are circular or elliptical areas varying in size from a small pea to a pin's head. They are greyish-white in colour, with a lightly raised periphery, depressed centre, and no surrounding redness. The papillary lines of the fingers are no longer visible even with a lens, the affected skin resembling parchment. Such marks may be readily overlooked, since they are absolutely painless and always heal perfectly if left alone.

Changes which are similar in type, but of a severer degree, result from a greater discharge of electric energy, the lesion being more extensive in surface area, and involving the subcutaneous tissues and possibly even the underlying muscles and bone. Such a condition is known as an electric necrosis, and when uncomplicated

by a superficial burning, due to the generation of heat by the current, is also painless.

There is no reaction or redness to be seen around the wound, the necrotic area being limited by a sharp outline. There are no evidences of suppuration, and the patient exhibits none of the toxic signs of a victim who has been burned; for the temperature remains low, the appetite is good, and the general condition is satisfactory. No contraction deformities are later to be seen, the wound healing slowly, and the necrotic area becoming covered with a new, soft and freely movable skin.

These characteristics apply only to the pure electric lesion, and are often masked by burnings due to the mechanical effect of the heat of the current. The lesions are then painful, and the usual toxic signs which accompany burning are present. It is, indeed, safe to say that where an electric lesion is painful and constitutional disturbances are observed, a certain amount of pure burning has complicated the electric lesion.

Areas of pure electric necrosis tend to enlarge for the first three weeks, both superficially and deeply, much of the dead tissue being shed. The third week is the danger period, for severe hæmorrhages may occur owing to degenerative changes in the artery walls. Should the nursing staff be unprepared for this contingency, the hæmorrhage may be so rapid and so severe as to result in death.

Treatment.

Conservative treatment is the golden rule which has now superseded the old idea of amputating the affected limbs. Experience has shown that a wonderful healing power is a characteristic of electric necrosis, and that surgical interference may result in severe septic complications. The wound should be dressed with antiseptic vaseline dressings and kept at rest, any deformity of the limb being corrected by the use of a light splint. Hæmorrhage occurring in the third week is treated by compressing the supplying artery proximal to the area of necrosis. Immediate surgical interference is only indicated when the affected limb is a source of toxins which circulate in the blood stream and threaten the life of the patient. These cases are fortunately rare.

Case 2.—Trost, aged fourteen. An example of a pure uncomplicated electric lesion. On May 6th, 1933, he was playing on the roof, when he grasped hold with both hands of two conduct

wires which were carrying the current to the house for lighting purposes, and was electrised by 220 volts alternating current. These wires were mounted contrary to all regulations, less than two metres above the roof. He did not lose consciousness, and felt no pain, and his general condition was excellent; but the right hand suffered a severe electrical necrosis extending from the distal crease in the palm to the finger tips. In the palm, where the conductor had been tightly grasped, a deep impression was visible. The skin over the injured area was torn and mutilated, leaving a necrotic area underneath. The left hand was slightly injured with small scattered necrotic areas over all the fingers (see Fig. 2). This was a pure electric lesion of the more extensive type with necrosis. It is interesting that, although the lesion seemed so severe, the patient experienced no pain either at the time of the accident or later. The general condition was, therefore, unaffected, the patient having no increased pulse rate, no temperature, and an excellent appetite.

He was put to bed for a few days, and the wound dressed for the first five days with a greasy antiseptic ointment. On the sixth day a benzene compress was applied for about fifteen minutes, and this was repeated daily for the next five days. This induces an intense hyperæmia, and it was hoped thereby to speed up the process of necrosis so that the tendons would not lie exposed for so long. On the tenth day following the accident the patient felt a warm feeling over the right hand, and saw that it was bleeding. He had already been warned of this possibility, and at once called the sister. The hæmorrhage was at once controlled by a tight compression bandage, but six hours later it began again, and was once more stopped, this time permanently. There is every likelihood that this hæmorrhage was caused by the hyperæmia following the benzene treatment; this was therefore stopped, as it had already served its purpose. At this time the general condition was still excellent. On the right hand the mutilated skin had all been shed, and under this, over an area a little wider than that originally described, was an extensive necrosis reaching deeply to the bone. This area showed the typical sharply-marked outline, and there was no trace of any surrounding reaction. The joint between the distal and middle phalanx had been opened as a result of the necrosis, but there was no sign of any sepsis in the joint. The distal end of the middle phalanx could be seen protruding from the

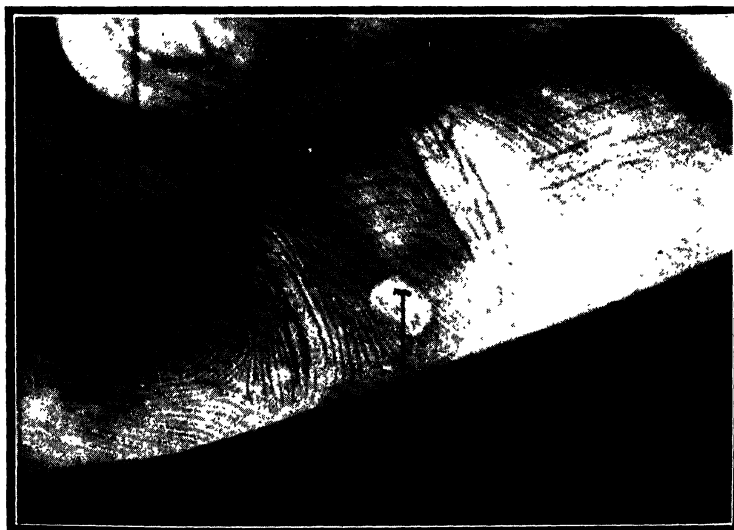


FIG 1 —A typical electric mark. A—Elliptical area with raised periphery and depressed centre. Note the absence of surrounding reaction.

A

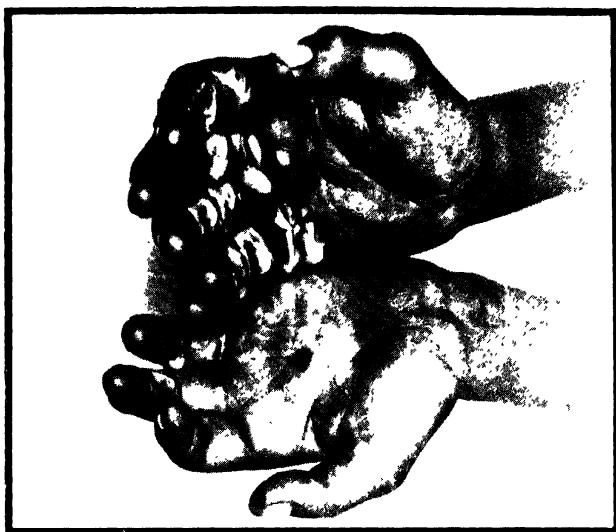


FIG 2 —Case 2 —Troost, aged 14. Photograph taken on admission, showing severe electrical necrosis of right hand, and much slighter injury to left. The black spot on palm of left hand is a wart.

"CLINICAL FEATURES AND TREATMENT OF ELECTRICAL INJURIES," by
Hilary Long, B.Sc., M.B., B.S.

To face page 394

destroyed joint, at first covered by normal bluish-white cartilage, but later, at the end of another week, the exposed bone appeared dead, the end being yellow in colour and dull to percussion with forceps. There was no pain on touching the exposed bone, and X-ray at this stage was negative. A wonderful healing process now set in. The hand was mounted on a light wire splint, and the fingers, which showed a slight tendency to a flexion contraction, were lightly bound out on the splint in a position of extension. The wound was dressed daily, and a perfect healing was effected.

Group (c)

One must not presume on the early and complete recovery of a patient from an electric accident, for there are certain effects which are not visible to the clinical eye, and may only appear after an interval of some hours; the patient apparently recovering from the general functional disability, and then complaining of a more or less localised pain. The most important lesions of this type occur in bones, peripheral nerves and some of the internal organs.

The passage of electric energy through a long bone which lies in the path of the current may give rise to a fine crack or schisis in the bone which is zig-zag in form, and is at first only visible microscopically, the X-ray taken immediately after the accident being completely negative. Such a lesion is always characterised by a localised pain in the limb of the affected area. This schisis may heal immediately and never be visible in the X-ray plate, but usually at the end of from three to six weeks the crack becomes wider and becomes visible radiologically. Healing most frequently occurs in the course of from six to twelve weeks, without obvious callus formation; but if the lesion is a severe one, the piece of bone affected may become separated and form a sequestrum, which must be removed not earlier than a year later by open operation.

A peripheral neuritis is another change which may follow the passage of an electric current through a limb. This appears within one or two weeks after the accident. The paralysis which follows is usually only partial, and there is in most cases a slight accompanying muscular atrophy. The prognosis is good, the muscular power or sensation returning in the course of two or three weeks. Thrombosis of vessels of the liver or kidneys is sometimes seen, and a temporary jaundice or hæmaturia may follow an electric

trauma. In these cases the organs affected are sufficiently large to withstand a small localised ischæmia, and no permanently bad results follow. Functional disturbances of the blood vessels, causing a spastic contraction or vaso-dilatation, may also occur in the internal organs, but similarly do not endanger the life of the victim. Should, however, the vessels of the brain or the coronary vessels of the heart be affected the result may be fatal.

Treatment.

If schisis of a bone is suspected the limb should be kept at rest in good position in a light plaster. An X-ray should be taken immediately, and repeated at regular intervals, the course of the case being followed in this way. Further treatment is usually unnecessary, as union readily follows. Jaundice or hæmaturia often clears up quickly. Should a brief period of anuria follow the electric trauma, spasms of the vessels of the kidney must be suspected, and local compresses of benzene to the lumbar region are usually followed by restoration of function.

Case 3.—Zlatoklanek, aged thirty-five. This case is an example of one where the lesions are only internal. On May 6th, 1933, while working on a mast, patient came too near the bare conductor wire, and was struck on the left arm with 10,000 volts and rendered unconscious for forty-eight hours. Following the recovery of consciousness on the second day he had a severe hæmoptysis. This was followed on the seventh and fifteenth day by further hæmoptyses. This is a very rare complication of an electrical accident. After the third bleeding the patient was sent to the Allgemeine Krankenhaus, in Vienna, for observation. On examination we found that the patient was a strong, muscular man. There were signs of Horner's syndrome on the left side, *i.e.*, there was ptosis, enophthalmos and a constricted pupil. This indicates damage to the left cervical sympathetic. We also found that there was an ulnar nerve paralysis on the left side.

How are we to explain these scattered clinical findings of hæmoptysis, cervical sympathetic damage and ulnar paralysis? Can we find a common cause for all three? We believed that the symptoms were due to a central nervous system lesion in the lower cervical region of the spinal cord in the neighbourhood of the centrum cilia spinale. It is possible for a lesion here to affect the fibres which form the ulnar nerve as they leave the spinal

cord, also the fibres passing to the sympathetic, and to catch a few fibres of the left vagus nerve. Probably the hæmoptysis was produced by this damage to the vagus, causing arterial injury and leading to a small gastric erosion. An X-ray was taken of the lower cervical vertebræ to exclude any possibility of bone injury. This, as we expected, was negative.

The patient was treated at first by complete rest. As he developed no further hæmoptysis, he was given benzene compresses for the left side of his neck. He soon showed signs of improvement, and was able to leave hospital at the end of two weeks. This is a good example of a case where the lesion is entirely internal. The prognosis is good, and such patients usually make a complete recovery.

BIBLIOGRAPHY.

- "Electrische Verletzungen": S. Jellinek, Vienna.
- "Death by Electric Currents and by Lightning.": A. J. Jex-Blake, *B.M.J.*, I, 1913.
- "Electropathology": R. H. Jaffe, Chicago.
- "The Effects of Lightning, with special reference to the Nervous System": M. Critchley, *Bristol Med. Chir. Journ.*, 1932.
- "Neurological Effects of Lightning and Electricity": M. Critchley, *The Lancet*, January 13th, 1932.

CHAPTER LV.

COMMON FUNGUS INFECTIONS OF THE SKIN

BY

C. HOWARD WHITTLE, M.A., M.D., M.R.C.P.

Physician to the Skin Department, and Clinical Pathologist,
Addenbrooke's Hospital, Cambridge.

A FAMILIARITY with the commoner fungus infections of the skin is important, both because of their comparative frequency in general practice, and also because of all skin diseases they are the most readily amenable to treatment. Difficulties lie mainly in diagnosis, and once the diagnosis has been established there is nowadays little excuse for protracted or unsuccessful treatment. I propose in the present article to deal with them in the order of their importance, and to bring out those points which most rapidly establish the diagnosis, and I shall briefly indicate the appropriate treatment. Ringworm, of course, heads the list.

Ringworm of the Scalp.

It is well known that ringworm of the scalp is confined to children, and tends to disappear spontaneously at puberty, even in a case that remains untreated. The disease, which is highly infectious, starts as a circumscribed patch of scurf in which broken hairs appear early. Whitfield maintains that any marked scurfiness in a child's head should be regarded as ringworm until the most careful search and microscopic examination has proved it to be otherwise. This axiom is well worth remembering. Infected hair stumps can often be detected more readily if a little chloroform is dabbed on the patch; this causing them to appear granular and "frosted." Broken hairs should be sought for, and if found the diagnosis can be established by microscoping in liq. potassæ, in which they should be soaked for ten minutes. The spores are readily visible as a mosaic of round, highly refractile bodies, regular

in size and structure, and situated in and around the hair. The only bodies likely to cause confusion under the microscope are red blood corpuscles and oil globules. The former are present in scanty numbers, unless violence has been used in removing hairs, and should offer little difficulty: they are much less refractile than the spores. Oil globules, on the other hand, may be numerous, but vary very greatly in size, from tiny particles up to large droplets. For those who have not the time or the facilities for microscopy, a suspicious case is best sent to a dermatologist, since treatment will have to be carried out by a specialist in most cases.

While dealing with the subject of diagnosis there are two points I would stress. Confusion may arise between a patch of dandruff or psoriasis and ringworm, and between alopecia areata and ringworm. In dandruff and psoriasis there are no broken hairs, and in alopecia areata the broken hairs are typically shaped like an exclamation mark (!), and do not show the corrugated frosted appearance of ringworm hairs when treated with chloroform, neither do they contain spores. Difficulty most often occurs when the bald patch is scurfy. The differentiation is important, since complete epilation as a cure for alopecia areata is hardly the treatment one would choose; whereas for ringworm it is the only really reliable measure. The point is not one of mere academic interest. I have had several cases of alopecia in children sent to me as ringworm for epilation.

There is one form of ringworm of the scalp, "black dot" ringworm, which is very apt to be mistaken for alopecia areata, but as it is rare the difficulty does not often arise. In this form the bald patch is not scaly; but the hair follicles are not empty, as in alopecia, and contain the remains of a hair broken off very short, and usually black in colour. This hair contains the fungus: a large-spored endothrix, not the microsporon.

The other point in diagnosis which I wish to stress is the immense help afforded by the use of a blue filter on an ultra-violet light lamp for showing up hairs infected with ringworm. In this light the hairs fluoresce brilliantly. I have been able to track down a carrier of ringworm in a school by this means when other measures and methods of examination had failed; and while epilation is taking place, it is my practice now to examine the cases each week to see that all the infected hairs fall. Even a single infected hair can be detected in a head of hair otherwise healthy. A number

of clinics now use the lamp as a routine. Diagnosis is certain, and is made with great rapidity.

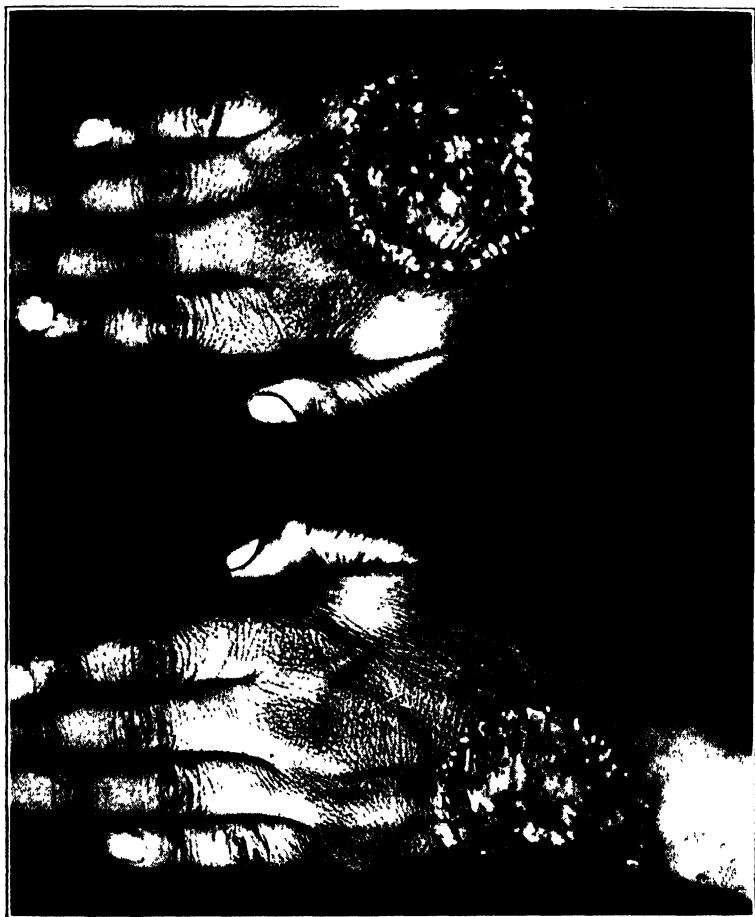
A form of scalp ringworm in which these filtered ultra-violet rays are not, however, of much service in diagnosis is the boggy, indolent, pustular swelling known as kerion. This is commonly an inch or so in diameter, purple in colour, with pus exuding from deep indentations—the hair follicles—the hair itself being usually absent. The inexperienced may be strongly tempted to put a knife into this in order to release pus from what in some ways appears to be an abscess. But the pus is not free, and incision will be useless; moreover, the swelling and inflammation are the natural means of cure, and no treatment is as a rule needed beyond encouragement with 1 : 4,000 perchloride of mercury compresses.

For the forms of ringworm of the scalp other than kerion, dermatologists are agreed that X-rays are the most certain and most rapid means of producing epilation, which is essential before the disease can be eradicated. Other methods of epilation, with the exception of thallium acetate by the mouth, are, in Whitfield's words, "dirty, painful and tedious," and if they are to succeed, require special skill in their carrying out.

Nowadays, with the aid of the filtered ultra-violet light, it is possible to define accurately the extent of the disease, and epilatory doses of X-rays can therefore with safety be limited to the affected parts provided the case is followed up and watched for two months. I believe in the efficacy of applying an antiparasitic ointment during the fall until all the hairs have fallen: it prevents loose fragments of hair falling about on to clothes, etc., and inhibits the growth of fungus on the scalp surface. During the fall, white cotton caps should, however, be worn in addition, and washed every other day. The ointment I use is Whitfield's:

Ac. benzoic	gr. 25
Ac. salicyl.	gr. 15
Paraff. moll.	dr. 2
Ol. cocois nucifer.	ad oz. 1

With regard to thallium acetate, many workers have used it and found it efficacious in many series of cases. I was unfortunate in my first trial of this drug, since the child was certainly not well during the fall, going off its food and looking pale and wan. I have employed it in a few cases since, and always with success. (It is given in a single dose, and the amount given is in strict pro-



Cattle ringworm affecting the hands (Photo by Dr. M. Sydney Thomson)

'COMMON FUNGUS INFECTIONS OF THE SKIN,' by C. Howard Whittle, M.A.,
M.D., M.R.C.P.

portion to the child's weight.) The indications are in those cases where X-rays cannot be given owing to the early age of the child (under four or thereabouts), or where for other reasons the child cannot be induced to keep still during the epilatory dose. In spite of these successes, I dislike using the drug owing to its undoubtedly poisonous nature.

Ringworm of the Glabrous Skin.

The difficulty in eradicating scalp ringworm depends upon the position of the fungus within the hair follicle and shaft. Drugs will not reach the spot in sufficient strength to kill the fungus without seriously damaging the skin. When ringworm occurs on the open skin the problem is usually a much simpler one.

Clinically, there are four chief forms of ringworm met with on the skin. The first, *tinea circinata*, is a *microsporon* ringworm which occurs on the skin of children who are, almost without exception, the subjects of scalp ringworm. The rings are pinkish in colour, slightly raised and finely scaly, are usually not more than an inch in diameter, and occur on the neck, shoulders, face, and sometimes on the arms. The scales contain the fungus. There is a large form somewhat similar, but which is not caused by the *microsporon*, and occurs independently of scalp ringworm; this is less common.

Another form which is seen quite commonly in agricultural communities, and which is the predominant form seen in the out-patient department in this district, is the rather heaped-up, indolent, oedematous circular patch which occurs on the face, neck, arms and hands of workers who tend cattle. This, by its firm, raised, indolent, usually painless character, often suggests an infective granuloma such as a gumma, or even in some cases actinomycosis. Usually it is possible to trace the follicular origin, and pus that may be expressed comes from the lanugo follicles. In this or the lanugo hair it is possible to find the fungus, a large-spored type, of which the spores are oblong and are usually arranged in chains. The diagnosis is easily made once the possibility has entered the mind of the practitioner, and those practising in agricultural districts soon acquire a familiarity with it. A history of association with cattle gives the cue. I have occasionally seen this form on the arms and legs of children, and it is possible that infection takes origin from fences and gates on which the children play, and on

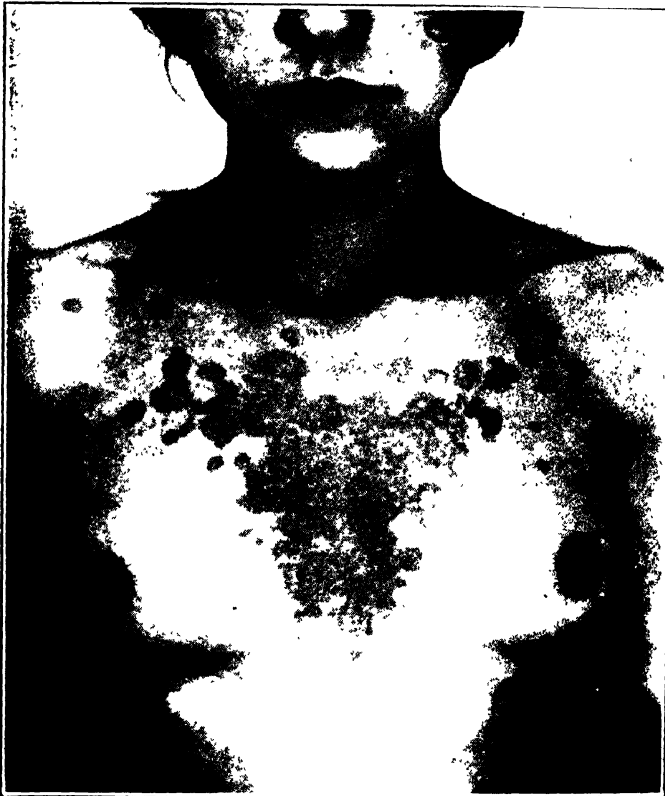
which cattle may frequently be seen rubbing themselves: at least this seems to me a likely explanation in the absence of a history of direct contact with infected animals.

A third important form which has been much more prevalent since the War is *tinea cruris*, or Dhobi's itch. This form, occurring in the groin and perineum as sharply marginate, scaly, itchy erythematous patches, is also common in Cambridge, but for quite another reason. It appears in undergraduates, who usually bring it with them from the public schools. It is a highly contagious disease, and often spreads widely in schools. Clothing, towels and baths are probably the means of spread, and boys are apt to be careless in these matters. It is not, however, confined to boys and young men, but may occur in persons of all ages. I recently saw a case in a lady, eighty years of age, which had puzzled her doctor for some weeks. Infection in such a case is possibly *via* a lavatory seat.

The older name for this form was *eczema marginatum*. It may present a raw, weeping surface, instead of the more common, dry scaly characters, but the important feature leading to a diagnosis is the sharply defined marginate nature of the lesion. It may be several inches in diameter. The scales show an abundance of the fungus in the form of a long, branching, fairly stout mycelium. Liq. potassæ preparations show it well.

A fourth form caused by the same organism is the intertriginous ringworm found between the toes, causing a sodden, reddened, scaly, sharply marginate lesion running a slow and chronic course. This form also appears to be increasing in frequency, and in America investigations indicate that spread is from person to person in the footways of swimming baths. This type usually affects the cleft between the little toe and the next, and may spread to adjacent clefts and involve the distal part of the foot, both dorsum and sole. It frequently remains localised to one foot, and this feature, when present, is in favour of the diagnosis, and against a non-infective eruption. The mycelium can usually be found without much difficulty in the scales. The same fungus may cause a vesicular eruption of the hands not unlike *cheiropompholyx*. The fungus can be demonstrated in the roofs of the vesicles.

The occurrence of generalised eruptions, secondary to the local affection, and known as *trichophytides* or *epidermophytides*, has been noted in recent years. One form, very like *lichen spinulosum*,



A typical example of pityriasis versicolor. The colour of the lesions at once distinguishes them clinically from a seborrhoeic eczema

(Photo by Dr. M. Sydney Thomson)

"COMMON FUNGUS INFECTIONS OF THE SKIN," by C. Howard Whittle, M.A., M D., M R.C.P

occasionally occurs in connection with epidermophytosis of the feet. An undergraduate came to me for the treatment of a patch of horny follicular spines appearing on the extensor aspect of the forearms, and it was not until I made special inquiry that I discovered a lesion of the toe-clefts, which examination proved to be due to ringworm. The treatment of the latter undoubtedly played a part in clearing up the trouble on the arms.

The treatment of body ringworm is simple, and most lesions clear up in a week or so under either 1:4,000 mercury perchloride compresses, which are specially suited to the heaped-up cattle types, or Whitfield's ointment for most of the others. Toe ringworm may require double strength ointment, and it is important to clip away the frayed scaly edges, and keep the toes separated. More resistant cases of toe ringworm will respond to 1:2,000 warm baths of potassium permanganate twice daily (Whitfield). Ultra-violet light is cumbersome, and usually unnecessary, for treating body ringworm. Simpler methods are to be preferred.

Ringworm of the Beard.

This commonly manifests itself as a circumscribed boggy patch on one side of the face, with some pustulation in the follicles. The fungus, which is a large spored type, can be found in the pus or hair stumps. The differential diagnosis from sycosis of staphylococcal origin presents certain difficulties. The latter is less heaped up, less boggy, and less frankly pustular, and runs a more chronic course. Usually beard ringworm will yield to mercury perchloride compresses. X-ray treatment will cause resolution in resistant cases.

Ringworm of the Nails.

One or more nails on one hand may be affected, and the nail is much coarsened, longitudinally ridged and thickened. Scrapings must be soaked twenty-four hours in potash before the fungus is visible under the microscope. X-ray treatment offers the most rapid method of cure, but some cases may be very resistant to treatment.

Tinea Versicolor.

This condition shows as a yellowish-brown discoloration of the skin, usually the trunk, with a sharply defined edge. It may extend in a sheet across the trunk, or may occur in patches only.

The lesion is not visibly or palpably raised, and may not even be scaly.

I have had patients referred as cases of Addison's disease, on account of the pigmentation, but the discoloration is all on the surface of the epidermis, and if the horny layer is scraped off, the fungus, *microsporon furfur*, is found in abundance as mycelium, club-shaped bodies, and spores. The condition is not very infectious, as a man may have it for years and not give it to his wife. It clears up readily with Whitfield's ointment.

Monilia Infections.

Apart from thrush, there are now known to be other lesions associated with monilia infections. The commonest is probably a bolster-like, œdematous swelling of the nail fold—paronychia. The condition is painful, non-purulent, and runs a chronic course. The yeast-like fungus can be found fairly readily in scrapings from beneath the fold. Treatment consists in applying 10 per cent. monsol daily on a match-stick swab. X-rays will cure resistant cases. Monilia may also give rise to intertriginous eruptions in moist places, such as the axillæ, groins and under the breasts. It is as well to remember that the presence of the fungus in a lesion does not necessarily imply that this organism is the cause ; it may be merely a secondary invader.

I have endeavoured to stress only those conditions which are most likely to be met with in general practice. I have purposely left out such infections as favus and erythrasma, which are at present uncommon. I have also left out the "seborrhœic" group of infections, which, though they are due in part to an organism of the fungus type, the *pityrosporon* of Malassez, are also associated with other skin organisms, notably the *acne bacillus* and the *staphylococcus albus*. Only by limiting the field of discussion is it possible in a short paper of this sort to deal even briefly with the commoner fungus infections of the skin.

CHAPTER LVI.

PIGMENT FORMATION IN THE SKIN

BY

G. H. PERCIVAL, M.D., Ph.D., F.R.C.P., D.P.H.

Lecturer on Diseases of the Skin, Edinburgh University; Assistant
Physician to the Department for Diseases of the Skin, Royal
Infirmary, Edinburgh.

THE pigment of the skin is a complex chemical substance known as melanin. This coloured substance is widely distributed throughout the animal kingdom, and although in the lower animals other pigments are found, melanin is the most universally distributed. In human beings under various pathological conditions, pigments other than melanin may be present in the skin, though melanin is the only one occurring normally and which is actually formed by the skin. Melanin is also found in the cells of the retina, choroid, ciliary body, iris, and in the substantia nigra.

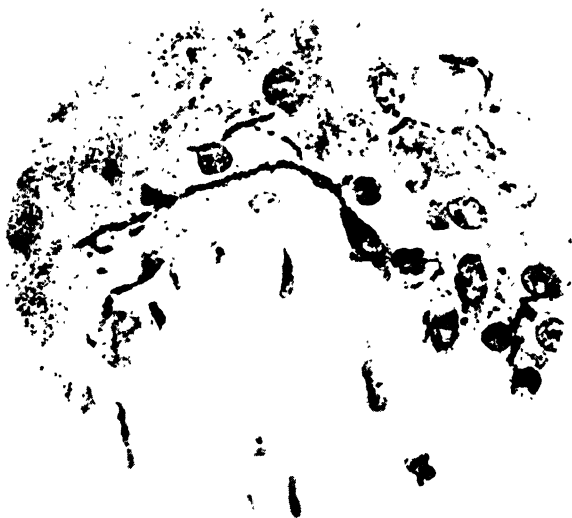
Within recent years our knowledge of skin pigmentation has advanced considerably. It has been shown that melanin formation takes place in pieces of skin removed from the general circulation, thus proving that the pigment is actually formed *in situ* within the cells in which it occurs. More recent work has shown that it is produced by the action of a ferment on a colourless substance.

In the skin melanin is found in the lowest layer of the epidermis and in the cells of the hair matrix. It occurs as fine, light-coloured particles, which are massed in the upper part of the cells, forming a cap overlying the cell nucleus. In white human skin all the basal cells do not contain pigment, and the amount of pigment varies with the situation and also as a result of exposure to certain stimuli, the most potent of which is ultra-violet light. The pigment content is greatest in the skin of the nipple, axilla and scrotum. In the dark races all the basal cells contain a greater or less amount

of melanin granules. When special staining methods are used to show up the melanin content of the cells more clearly, it is found that some of the pigment-containing basal cells possesses finger-like processes which stretch upwards and pass into the spaces between the overlying cells of the rete mucosum. These dendritic cells, as they are called, are most numerous in highly pigmented areas. For a time they were confused with other dendritic cells present in the skin. The latter are nerve elements, and special staining methods clearly differentiate these two types of cell, which are so different functionally. When pigment formation is being actively engaged in as a result of stimulation, the dendritic cells increase in number, and it is highly probable that all basal cells are capable, under certain circumstances, of assuming a dendritic form. Dendritic cells are found all over the skin surface, and also in the mucous membrane of the mouth and pharynx.

Returning to the chemical aspect of the problem, it has been mentioned that melanin is formed by the action of a ferment on a colourless substance, and that this action takes place within the living cells. The evidence for this statement is indirect, neither the ferment nor the chemical on which it acts having been isolated. It has been found, however, that when a section of skin is treated with the chemical dioxypyphenylalanine and then examined, the pigment content of the basal layer of the epidermis has increased. The cells contain a dark substance indistinguishable from melanin, and dendritic cells which were previously invisible make their appearance. This reaction with dioxypyphenylalanine is specific, and does not occur even with very closely related chemical substances. The reaction is inhibited by heat, by cyanide and by antiseptics, and does not occur in dead skin. These facts, together with its specificity, strongly suggest that the reaction is due to an oxidising enzyme which acts either on dioxypyphenylalanine, or on a closely related substance not yet known. It closely resembles the action of tyrosinase on the amino acid tyrosine, and it is believed by some observers that the reaction *in vivo* actually involves tyrosine and tyrosinase, although there is no direct proof for this.

Melanin-containing cells are found in the dermis, but histological experiments have shown that those normally found in that situation have not the power to form melanin. They are merely carriers of preformed melanin which they have apparently phagocytosed from the epidermis. These melanin-containing cells of the dermis



Dendritic cell in basal layer of epidermis (section stained by silver method)



This section of skin has been treated with di-oxy-phenyl-alanine. The pigment-forming ferment has oxidised this substance, within a number of basal cells, to a black melanin.

"PIGMENT FORMATION IN THE SKIN," by C. H. Percival, M.D., Ph.D., F.R.C.P.

are then phagocytic fibroblasts. There is every reason to believe that only epidermal structures have the power of forming melanin. The only possible exception to this statement is the blue nævus, or Mongolian spot. This is a bluish-black spot usually situated over the sacral region, which consists of a collection of pigmented cells situated deep down in the dermis. These cells are capable of forming melanin, and there is a controversy as to whether they are of epidermic or mesodermic origin. The pigmented cells of the ordinary nævus or pigmented mole have, of course, been definitely proved to be derived from the epidermis. Melanin-forming cells are called melanoblasts, while those which merely carry melanin are referred to as melanophores.

The formed melanin is ultimately disposed of in one of two ways: it may gradually reach the skin surface and be lost in desquamation, or it may be extruded downwards into the dermis, where it is removed by phagocytic fibroblasts. The pigment of the hair is formed in the hair matrix and incorporated in its fully-formed state in the upward-growing hair shaft. The reason for red hair probably lies in the distribution of the melanin granules in the cells of the hair shaft; chemically, the pigment seems to be identical with that of black or brown hair.

The chief function of melanin in the skin is to protect the underlying structures from the injurious effects of excessive exposure to ultra-violet light. In man this is its sole function, but in the lower forms of life, variations in colour play an important part in the preservation and propagation of the species. Exposure of the skin to ultra-violet rays produces two reactions in the epidermis. Pigment formation is stimulated. Basal cells which were in a resting stage as regards pigment metabolism suddenly become active, many assume the dendritic form, and in each case melanin granules are formed. These are massed above the cell nucleus as if to protect it from further radiation. In this process it can be demonstrated that the first step is an increase in the content of melanin-forming ferment in those cells which ultimately contain pigment. This layer of pigment acts as an absorbing medium for the more penetrating ultra-violet wave-lengths.

Freckles consist of a localised increase in the number of pigmented cells in a given area of the basal layer, combined with a relative increase in the amount of melanin contained in the individual cells.

In order to protect the cells of the rete mucosum which lie above this layer of melanin-containing cells, another mechanism is brought into play. Cell division in the basal layer is speeded up, and the chemical changes which ultimately produce the stratum corneum are hastened, so that in a very short time the stratum corneum becomes considerably thickened. This thickened horny layer then acts as a screen for the upper layer of the epidermis with their contained nerve endings. A third effect of ultra-violet light, but one which is not related to pigment metabolism, is the formation under the influence of vitamin D from chemical changes in the epidermal sterols. This reaction probably takes place in the region of the stratum granulosum of the epidermis, under the influence of wave-lengths in the region of $280\text{ }\mu\mu$.

Physiological variations in the pigment content of the skin occur in association with climatic conditions, advancing years, and under the influence of the endocrine adjustments of pregnancy. That associated with advancing years is brought about slowly, and affects the hair matrix, producing canities. The amount of pigment-forming ferment in the matrix cells gradually diminishes and finally disappears altogether, and during this process a considerable amount of the pigment which is formed does not pass upwards in the hair shaft, but is extruded into the dermis and phagocyte.

Changes in temperature can exert considerable influence on pigment metabolism. This effect is, however, not obvious in the human, but is very well shown by the Himalayan rabbit. This animal's fur is white, except for that on the tip of the nose and on the paws. If an area of skin covered with white fur is shaved, and the surrounding temperature kept low, the fur grows in black on the shaved area. As the hair increases in length and forms a warm covering for the area, pigment ceases to be formed, so that the fully-grown hair consists of a white shaft having a black tip.

Abnormal pigment formation is seen in all forms of endocrine disturbance, and may take the form of over-production of pigment, either in localised areas or generally all over the body, or else in localised or generalised loss of pigment. The latter condition, when temporary and localised, is termed leucoderma; when permanent and localised, vitiligo. In both these conditions the cause of the loss of pigment is the same, namely, loss of the ferment. This state of affairs exists in albinism.

Gross disturbance of pigment metabolism occurs in Addison's disease. The increased pigmentation can be satisfactorily explained on the basis of the view of melanin formation which has been outlined above. The colourless precursor of melanin must be closely related to the immediate precursor of adrenaline. In Addison's disease adrenaline formation is much diminished on account of the degenerative changes in the suprarenal glands, so that an excessive amount of the chemical precursor of adrenaline accumulates in the circulation. This excess is acted on by the melanin-forming ferment of the skin cells, and converted into melanin with a resulting hyper-pigmentation. This view is strengthened by the finding that in Addison's disease the ferment is present in the skin cells in diminished amount, suggesting that it has been depleted as a result of acting on an excessive amount of pabulum.

Excessive pigmentation frequently occurs in skin which has been the seat of inflammation, and is purely secondary to the underlying vascular congestion. Certain types of inflammatory lesion are more prone than others to be followed by inflammation, and in this respect syphilis is conspicuous. Hyper-pigmentation is caused by X-rays, radium and thorium radiations, the pigment-forming cells being stimulated in a manner analogous to that seen with ultra-violet rays.

Pigmented tumours of the skin are met with in almost everyone. They are usually referred to as *nævi* or melanoma. At one time there was considerable controversy as to their derivation, but it has been shown by numerous workers that the pigmented cells which are characteristic of these new growths are of epidermal origin. The tumours are composed of masses of pigmented cells situated high up in the dermis. These cells have literally dropped from the epidermis into this situation, and have then multiplied. The tumours vary in size from small pin-head, brownish points, to warty, pigmented growths which cover a large area of skin. The more extensive growths sometimes have a linear arrangement, and the condition is referred to as *ichthyosis hystrix* or *nævus unius lateris*. The large growths are usually present at birth, but the smaller variety often first appear during childhood, or even adult life. Frequently in the larger growths the pigmented cells are associated with an extensive development of hair follicles. A small percentage of these pigmented *nævi* become malignant, and it is the bluish-black type situated on the hands and feet which do

so most frequently. The nævus first enlarges, then satellite nævi appear in the neighbourhood, and very soon the regional glands may become enlarged. Spread also takes place by the blood stream, and metastases occur in the skin, lungs and liver. The condition is rapidly fatal. Sometimes pigmented nævi appear for the first time late in life, and this type frequently develops malignant features. Malignancy may be stimulated by local irritation and by incomplete removal of an apparently benign tumour. In view of this, any method of removal of such lesions for cosmetic or other reasons should be thorough and complete.

INDEX

- Abdominal conditions in children,
acute, 337.
 Blood transfusions in, 343,
 347.
 Causes of, 338.
 Prognosis, 347.
 Saline injections in, 343, 347.
 Symptoms and signs, 339.
 Treatment, 342.
 Operations in children, after-
 treatment, 347.
 Operative details, 344.
- Abscess, "collar-stud," in the palm,
208.
 in Hip disease, 301.
- Acarine mange from cats or dogs, 326.
- Achlorhydria, 370.
- Acholuric family jaundice, 50.
- Acidosis in jaundice, 123.
- Acriflavine solution, 117.
- Adalin, in insomnia, 188.
- Adams-Stokes attack, 11.
 Syndrome, 12.
- Addisin, in pernicious anæmia, 246.
- Addison's anæmia, 239, 372, 409.
- Adenoid hypertrophy, 189.
- Adexolin in pink disease, 264.
 in Subacute combined degenera-
 tion of the spinal cord, 245.
- Adrenal obesity, 28.
- Adrenalin in Adams-Stokes attack, 12
 in Diabetic coma, 6.
 Intracardiac injection, 12.
- Adrenalin in hay-fever, 145.
 in Peripheral failure, 10.
 Pink disease, 264.
 Prolonged faints, 10.
 Syncope, 14.
 Vaso-vagal attacks, 10.
 Intracardiac injection of, 12.
- Aerated sitz-bath, 356.
- Agglutination tests, 65.
- Agrypniaphobia, 183.
 Treatment, 186.
- Aix method of muscle massage, 306.
- "Aklorop" tablets in hay-fever, 145.
- Albinism, 408.
- Alcohol for bronchopneumonia in
children, 296.
- Alkalies in liver cases, 123.
- Alkaline baths for pruritus, 124.
 Water at Cheltenham Spa, 363.
- Allergy, hay-fever, 139.
 Pollen, 137.
- Allonal in insomnia, 188.
- Almata in pink disease, 261.
- Ambulatory treatment, leg fractures,
353.
 in Hip disease, 300.
- Ammonium bromide in insomnia, 185.
 Chloride in pyorrhœa, 165.
- Amnesia, 75.
- Amylene hydrate in insomnia, 188.
- Anæmia, achlorhydric, 366.
 Aplastic, 367.
 Addisonian, 372.
 Constitutional, 276.
 due to deficiencies, 366.
 Erythroblastic, 276.
 Ferri et ammon. cit. in, 371.
 Hog's stomach in, 372.
 Hyperchromic macrocytic, 366,
 370, 372.
 Hypochromic microcytic, 366,
 369.
 in infancy, copper in, 275.
 Dietary factors, 273.
 Hygienic factors, 273.
 Infections, 273.
 Orange juice in, 273.
 Transfusion in, 275.
 Treatment of, 271.
 Lack of iron in, 272, 275.
 Liver extract in, 372.
 Mediterranean, 275.
 Nutritional, 366.
 Pernicious, a deficiency disease,
 245 (*see* Pernicious anæmia).
 Addisin in, 246.
 Nervous symptoms, 240.
 of the newborn, 276.
 Prematurity, 271.
 Physiological, 271, 276.
 Post-hæmorrhagic pernicious, 366.
 Splenic, 49.
 von Jaksch's, 276.
- Anæsthesia, chloroform, death in, 11.
- Anæsthetic, local (in tapping chest),
132.
- Anergia, 75.
- Angular conjunctivitis, zinc sulphate
for, 289.
- Anisocoria, 87.
- Ankle-joint fractures, bone traction
in, 354.
 Delbet splint, 353.
 Dupuytren's, 349.
 Immobilisation in plaster of
 Paris, 351.
 Kirschner's wire transfixion pin,
 354.
 Open operation, 355.
 Pott's, 349.
 Skin traction, 355.
 Stocking suspension, 355.
 Treatment by traction, 354.
 Wagstaff's, 350.
 X-ray of, 350.

- Antiphlogistine in rheumatoid arthritis, 201.
- Antitetanic serum in compound fracture of skull, 18.
- Typhoid vaccine in disseminated sclerosis, 55.
- Antrum, maxillary, empyema of, 225.
- Liquid paraffin for lavage, 226.
- Aolan in erysipelas, 281.
- Apathy, 75.
- Aphonia, functional, 381.
- Suggestion in, 382.
- Appendicitis, drainage in, 344.
- in children, 343.
- Jaundice after, 50.
- Operative details, 344.
- Argyrol in conjunctivitis, 287, 310.
- Arm lymphangitis, linseed poultice for, 207.
- Arsenic in disseminated sclerosis, 56.
- Pink disease, 259, 264.
- Pyorrhœa, 163, 165.
- Arteriosclerosis, insomnia of, 180.
- Arthritis, rheumatoid, 199.
- Artificial respiration in electrical injuries, 391.
- Sunlight in infantile convulsions, 148.
- Aspiration of empyema, 294.
- Hip abscess, 301.
- in pleurisy, 129.
- Aspirator, Dieulafoy, 132.
- Potain, 132.
- Aspirin in erythema nodosum, 269.
- in Pleurisy, 128.
- Rheumatoid arthritis, 201.
- Asthma, cardiac, 13.
- Hay-fever, 140.
- Ataxic paraplegia, 240.
- Atophan group in rheumatoid arthritis, 201.
- Atropine in biliary colic, 63.
- Burns and scalds, 21.
- Atropine in cardiac dyspnœa, 13.
- in Seasickness, 176.
- Aural discharge, syringing of, 18, 195.
- Mop, 197.
- Polypus, 196.
- Auricular flutter, syncope in, 11.
- Autohæmotherapy, for eczema, 329.
- Sero-therapy in pleural effusion, 134.
- Avoleum in subacute combined degeneration of the spinal cord, 245.
- Bacillus dysenteriæ, 220.
- Bacteriophage in furunculosis, 281.
- Baldness, X-ray, 327.
- Banana in coeliac disease, 252.
- Barbellion, disseminated sclerosis, 53.
- Basal fractures, skull, 18.
- Metabolism, individual, 27.
- Bath, Berthollet local steam vapour, 360.
- Bran, 360.
- Circular needle, 358.
- Fango, 360.
- Immersion, 357.
- Local, 358.
- Mud, 360.
- Mustard, 360.
- Pack, 360.
- Paraffin wax, 360.
- Peat, 360.
- Pool, 257.
- Spa, for rheumatism, 307.
- Sedative, 363.
- Thermal vapour, 363.
- Vapour, 360.
- Wilde thermal couch, 360.
- Beard, ringworm of the, 403.
- Bedsore in combined subacute degeneration of the spinal cord, 246.
- Bemax in subacute combined degeneration of the spinal cord, 245.
- Benedict test, 2.
- Bicarbonate sodium in nasal catarrh, 227.
- Bile acids in migraine, 92.
- Ducts, congenital obliteration of, 47.
- Biliary colic, atropine in, 63.
- Differential diagnosis, 61.
- Dilaudid in, 63.
- Evipan sodium in, 63.
- Morphia in, 62.
- Operation in, 63.
- Dyskinesia, 58.
- Binocular magnifiers in eye work, 105.
- Bismuth carbonate in diarrhœa, 220.
- in syphilis, 39.
- Blackley on hay-fever, 137.
- Blepharitis angularis, 319.
- Ciliaris, 319.
- Marginalis, 319.
- Epilation in, 319.
- Silver nitrate in, 319.
- Squamosa, 319.
- Ulcerosa, 319.
- Zinc sulphate in, 319.
- Blood groups, 64.
- Transfusion, 64.
- Air-lock in, 69.
- Citrate method, 66.
- for Hæmatemesis, 71.
- Hæmophilia, 71.
- Newly born, 71.
- Leukæmia, 71.
- Marasmus, 71.
- Melæna, 71.
- Peptic ulcer, 71.
- Pernicious anæmia, 71.

Blood Transfusion :

- for Purpura, 71.
- Rheumatoid arthritis, 203.
- Ulcerative colitis, 71.
- French's needle in, 67.
- Indications for, 70.
- London service, 68.
- quantity used, 71.
- Blue filter on ultra-violet light, 399.
- Nævus, 407.
- Bockhart's impetigo, 283.
- Body weight, normal, 26.
- Factors in the preservation of, 26.
- Boric lotion for conjunctivitis, 287.
- Bostock on hay-fever, 137.
- Boutall's cosmetics, orris-root-free, 142.
- Bowman's needle, in eye work, 106.
- Brachial neuritis, Spa treatment for, 304.
- Brain, modern methods of exposing, 331.
- Raw, treatment by, 244.
- Breaking of the voice at puberty, 381.
- Breast, massage of, 216.
- Milk, composition of, 213.
- Quantity of, 216.
- Overfeeding, 218.
- Sponging, 216.
- shy child, 214.
- Under-feeding, 213.
- Breast-fed infant, digestion in, 211.
- feeding, chloral in, 214.
- Green stools in, 213.
- Irregular, 213.
- Nervousness of child, 214.
- Night feeds, 213.
- Respiratory obstruction in, 214.
- " Snuffles " in, 214.
- Bridge of Allan Spa, sedative, 363.
- British Spas, cost at, 307.
- Ultra-violet light at, 306.
- Broderick, F. W., views on pyorrhœa, 161, 165.
- Bromides in epilepsy, 151.
- in Insomnia, 188.
- Seasickness, 173, 177.
- Bromocarpinæ, syrup, in epilepsy, 151.
- Bronchitis, acute, in children, 296.
- Broncho-pneumonia in children, alcohol for, 296.
- Creosote for, 295.
- Oxygen for, 295.
- “ S.U.P. 36 ” (B.D.H.), 295.
- Burke's method, syphilis, 41.
- Burns, atropine in, 21.
- Collapse in, 20.
- Morphia in, 20.

Burns

- Mortality from, 24.
- Routine treatment, 20, 22.
- Bursæ of hand, 208.
- Buttermilk in cholera infantum, 223.
- Butyrate of manganese in furunculosis, 281.
- Buxton, bracing spa, 363.
- for rheumatism, 307.
- Sycosis, 283.
- Buzzard, Farquhar, on disseminated sclerosis, 56.
- Caffeine, idiosyncrasy, 180.
- in migraine, 92.
- Rheumatoid arthritis, 201.
- Calamine lotion, 283.
- Calcium caseinate in ileo-colitis, 221.
- Chloride in infantile convulsions, 148.
- Deficiency in infantile convulsions, 148.
- in liver cases, 123.
- Lactate in migraine, 90.
- Pink disease, 261, 264.
- Levinulate, 123.
- Calculi, kidney, 120.
- Caloplast for children's pleurisy, 294.
- Camphor in oil, for children, 16.
- Campolan (liver extract), 244.
- Cancer of the larynx, 386.
- Radium in, 387.
- Carbohydrates in lichen urticatus, 329.
- Carbon dioxide in hay-fever, 145.
- Caruncle of the kidney, 118.
- Cardiac asthma, 13.
- Dyspnoea, acute, 13.
- Atropine in, 13.
- Failure, acute, 11.
- Digitalis in, 12.
- Intravenous glucose in, 12.
- with pain, 12.
- Cardiac syncope, 11.
- Cardiozol-ephedrine in peripheral failure, 11.
- Carotene, 30.
- Casec in ileo-colitis, 221.
- Casein in cholera infantum, 223.
- Cassowary neck, 34.
- Castle on pernicious anæmia, 245.
- Castor oil mixture in diarrhœa, 220.
- Cataplasma kaolin in pleurisy, 127.
- Catarrh, summer, 137.
- Catarrhal conjunctivitis, acute, 289.
- Argyrol for, 289.
- Zinc sulphate for, 289.
- Jaundice, 48.
- Catechu, tinct., in diarrhœa, 220.
- Catheter, St. Mark's apparatus, 4.
- Tying in, 4.

- Cautery in pyorrhoea, 165.
 Cerebral adiposity, 35.
 Contusion, vertigo due to, 101.
 Embolism, psychosis of, 79.
 Hernia, 333.
 Tumour, psychosis of, 79.
 Cervix, amputation of, 114, 116.
 Chalazion, 107, 309.
 Charcoal in subacute combined degeneration of the spinal cord, 247.
 Cheltenham, sedative spa, 363.
 Chemical burns, tannic acid in, 24.
 Chest, exploring, 131.
 Needling, 131.
 Tapping, 131.
 Exploration, novocaine in, 132.
 Percaine in, 132.
 Phenolaine in, 132.
 Child, rectal examination of, 341.
 Childbirth, insanity after, 76.
 Childhood, epilepsies in, 146.
 Children, appendicitis in, 343.
 Bronchitis, acute in, 296.
 Bronchopneumonia, creosote in, 295.
 Common skin diseases of, 323.
 Concussion in, 16.
 Convalescence, ultra-violet light in, 295.
 Cough, alkaline expectorant, 293.
 Dermatoses, sepsis group, 323.
 Diarrhoea, kaolin in, 293.
 Kaylene in, 293.
 Empyema in, 294.
 Eye diseases of, 316.
 Head injuries in, 15.
 Hernia in, 345.
 Herpes oticus in, 193.
 Insomnia in, 184.
 Lumbar puncture in, 16.
 Morphia to be avoided in, 16.
 Otitis media in, 189.
 Differential diagnosis, 193.
 Prophylaxis, 190.
 Treatment of, 194.
 Types of, 191.
 Warnings, 195.
 Pneumonia in, 291.
 Alcohol in, 293.
 Bismuth salicylate in, 293.
 Camphor in oil, 293.
 Coramine, 293.
 Lixen in, 293.
 Vitamin A and D (concentrate) 293, 295.
 Sedatives, 294.
 Chloral, 294.
 Nepenthe, 294.
 Paraldehyde, 294.
 Sinusitis in, 189.
 Skin disease in, 324.
 Chloral in breast feeding, 214.
 in Infantile convulsions, 147.
 Insomnia, 186, 188.
 Seasickness, 177.
 Chloretone in seasickness, 177.
 Common cold, 171.
 Chloroform anaesthesia, death in, 11.
 Cholera infantum, 221.
 Buttermilk in, 223.
 Casein in, 223.
 Strychnine in, 223.
 Cholecystitis, acute, differential diagnosis, 61.
 Pain of, 58.
 Chromic acid in pyorrhoea, 163.
 Chronic hoarseness, 379.
 Intestinal indigestion, 248.
 Rheumatism, Spa treatment, 303, 307.
 Circulatory disturbances, vertigo due to, 101.
 Failure, acute, 8.
 Cirrhosis, hepatic, acute, in infants, 49.
 Citrate method, blood transfusion, 66.
 Climacteric, insanity of, 76.
 Man's, 36.
 Clock, feeding baby by the, 213.
 Cocaine in nasal inflammation, 226.
 Coccil infections of the urinary tract, 118.
 Coccogenic syphilis, 282.
 Cocci, skin diseases due to, 278.
 Cod-liver oil in infantile convulsions, 148.
 Phlyctenular conjunctivitis, 289.
 Codeine as cough sedative, children, 293.
 Phosphate in rheumatoid arthritis, 201.
 Coeliac disease, 248.
 Banana in, 252.
 Biochemical signs, 250.
 Copper sulphate in, 253.
 Diagnosis, 251.
 Dietary treatment, 252.
 Eiweiss milch in, 252.
 Ergosterol in, 253.
 Fat Absorption in, 250.
 Ferri et ammon. cit. in, 253.
 "Force" in, 253.
 Marmite in, 253.
 Protein milk in, 252.
 Rickets, 249.
 Cold, common, chloretone in, 171.
 Glegg's mixture, 170.
 Jelopar in, 171.
 Local prophylaxis, 170.
 Menthol in, 170.
 Nasal douches in, 171.
 Parke, Davis' spray, 171.
 Pinoleum in, 171.

- Cold, common :
 Pneumonia after, 171.
 Sequelæ, 171.
 Sprays in, 170.
 Treatment of, 168.
 Vaccine therapy in, 169.
- Colitis, ulcerative, blood transfusion for, 71.
- Collip's placental hormone, in migraine, 91.
- Collosol argentum, pessary, 117.
 manganese in ecthyma, 284.
 in Furunculosis, 281.
- Colon lavage, 359.
 Normal saline in, 360.
 Pot. permang. in, 360.
- Colpoperineorrhaphy, Donald's, 115.
- Colporrhaphy, 114.
 Anterior, 115.
 Fothergill's, 115.
- Colsul in disseminated sclerosis, 55.
- Coma, diabetic, 1.
 Shock, in, 4.
 Sodium bicarbonate in, 4.
 Insulin, 2.
- Compral in migraine, 92.
- Concussion in children, 16.
- Condy's fluid for conjunctivitis, 287, 288.
- Congenital pyloric stenosis, 212.
 Rammstedt's operation for, 212.
 Hypertrophic stenosis of the pylorus, 232.
 Syphilis, 48, 379.
- Conjunctivæ, irrigating, 104.
 Painting, 105.
- Conjunctival test, Noon's, 137.
 Freeman's, 137.
- Conjunctivitis, argyrol for, 287.
 Boric lotion for, 287.
 Collyria for, 287.
 Condy's fluid for, 287.
 Lot. hyd. perchlor. for, 287.
 Koch-Weeks' bacillus, 310.
 Lavage in, 286.
 Morax-Axenfeld, 310.
 Oxycyanide of mercury for, 310.
 Phlyctenular, atropine in, 319.
 Allen & Hanbury's ung., 317.
 Calomel for, 289.
 Cod-liver oil in, 289.
 Ergosterol in, 319.
 Glycerin thymol co. in, 318.
 Glycerophosphates of iron in, 319.
 Iodide of iron in, 319.
 Syr. ferri iod. in, 289.
 Yellow oxide of mercury ung., 289, 317.
- Protargol for, 287, 288.
- Conjunctivitis :
 Silver nitrate for, 287, 288, 310.
 Tannic acid in, 288.
 Undine for, 287.
 Zinc sulphate for, 287, 289, 311.
- Convulsions, idiopathic of infancy, 147.
 in febrile states, 149.
 Infantile, 146.
 Luminal in, 150.
 Thyroid in, 150.
- Copper in anæmia of infancy, 275.
 Sulphate in coeliac disease, 253.
 Pyorrhœa, 164.
- Cornea, diseases of, 320.
 foreign bodies in, 312.
 Septic abrasions, 110.
 ulcers, 110.
- Coronary thrombosis, 12.
 Syncope in, 11.
- Cough sedative for children, codeine, 293.
 in pleurisy, 128.
 Heroin hydrochloride for, 128.
 Liq. morph. hydrochlor. for, 128.
- Cow & Gate separated milk, 221.
- Craniotomy, exploratory osteoplastic, 332.
 Skull plough for, 335.
- Creosote in broncho-pneumonia of children, 295.
 and alcohol in pyorrhœa, 163.
- Curettage in pyorrhœa, 165.
- Cushing's cross-bow incision, 334.
- Cyclitis, 315.
- Cyllin added to sulphur ointment, 326.
- Cystitis in subacute combined degeneration of the spinal cord, 246.
- Cystocele, 112.
- Dark glasses in hay-fever, 144.
- Davidson's fish-liver extract, 244.
- Davies and Gilchrist oxygen apparatus, 292.
- Decholin in migraine, 88.
- Delbet splint for ankle-joint, 353.
- Depersonalisation, 75.
- Depressed fractures in children, 17.
- Dermatitis, pollen, 140.
 Traumatic, 328.
- Dermatoses, children, sepsis group, 323.
- Dextrose in diabetic coma, 4, 6.
- Dhobi itch, 402.
- Diabetic coma, diagnosis, 2.
- Diarrhœa, bismuth carb. in, 220.
 Castor oil in, 220.
 Catechu in, 220.

- Diarrhoea :**
 Dyspeptic, 219.
 Fermentation, 219.
 in infants, 219.
 Inanition, 213.
 Kaolin in children's, 293.
 Kaylene in, 293.
 Normal saline in summer, 212.
 Summer, 212.
- Diathermy in rheumatoid arthritis, 202.**
- Diet in liver cases, 123.**
 in Rheumatoid arthritis, 203.
 Iron content of, 367.
 Salt-free, in obesity, 30.
- Dietary treatment of obesity, 30.**
- Digestive disturbance in jaundice, 124.**
- Digitalis, indications for, 14.**
 Poisoning, death in, 11.
- Dilaudid in biliary colic, 63.**
- Dimol in subacute combined degeneration of the spinal cord, 247.**
- Disorientation, 75.**
- Displacement method of Proetz, 229.**
 in nasal catarrh, 229.
- Disseminate sclerosis, 240.**
 Acute vaccinal type, 53.
 Ætiology, 51.
 and goitre, 52.
 Antityphoid vaccine in, 55.
 Arsenic in, 56.
 Barbellion's book, 53.
 Buzzard, Farquhar on, 56.
 Colsul in, 55.
 Diagnosis, 242.
 Electrical treatment, 57.
 Gowers on, 53.
 Hexamine in, 56.
 "Journal of a Disappointed man," 53.
 Marie, Pierre, on, 56.
 Massage in, 57.
 Mellanby on, 53.
 Mercury in, 56.
 Novarsenobillon (914) in, 56.
 Pot. iodide in, 56.
 Quinine in, 56.
 Silversalvarsan in, 56.
 Sulphosin Leo in, 55.
 Treatment, 51.
 Vertigo due to, 101.
 Vitamin A in, 56.
 Williamson on, 56.
- Donald, Archibald, 114.**
 Colpoperineorrhaphy, 115.
- Douche, 358.**
 Aix, 359.
 Fan, 358.
 Filiform, 358.
 Massage, 359.
 Scotch, 359.
 Vichy, 359.
- Dover's powder in pleurisy, 128.**
- Drainage by rubber tissue wicks, 210.**
- Drake & Thomson's course, syphilis, 41.**
- Droitwich brine spa, 363.**
 for rheumatism, 307.
- Dry chronic pleurisy, 128.**
 Cupping in pleurisy, 129.
- Duncan & Flockhart's pollen solutions, 142.**
- Duodenal ulcer, differential diagnosis, 61.**
- Dupuytren's fracture, 349.**
- Dyspareunia, 117.**
- Dystrophia adiposo-genitalis, 34.**
- Ear-plug, fibrous plasticine as, 198.**
- Easton's syrup in furunculosis, 281.**
- Ecthyma after scabies, 284.**
 Ultra-violet light in, 284.
- Eczema, autohæmotherapy in, 329.**
 Calomel in a zinc cream for, 329.
 Hog-spleen injections for, 329.
 Ichthyol for, 329.
 Lassar's paste for, 329.
 Marginatum, 402.
 Milk injections for, 329.
 Traumatic dermatitis and, 328.
- Effusions, pleural, 131.**
 Purulent, 135.
 Tubercular, 133.
- Eiweiss milch in cœliac disease, 252.**
- Elbon (ciba), in bronchopneumonia, children, 295.**
- Electric necrosis, 392.**
 Pads in rheumatoid arthritis, 201.
- Electrical injuries, 390.**
 Artificial respiration in, 391.
 Clinical features, 390.
 Horner's syndrome in, 396.
 Lumbar puncture in, 391.
 Treatment, 391.
 Disseminated sclerosis, 57.
- Electro-therapy in pyorrhœa, 166.**
- Electrolysis, in-growing eyelashes, 310.**
- Emetine in pyorrhœa, 164.**
- Emmenin complex (Glaxo), in migraine, 91.**
- Empyema, 135.**
 Aspiration of, 294.
 Drainage of, 295.
 in children, 294.
- Encephalitis lethargica and nasal catarrh, 227.**
 Sporadic, 148.
- Endocrine disturbance and pyorrhœa, 161.**
 Imbalance in psychoses, 77.

- Endogenous obesity, 34.
Entamoeba buccalis in pyorrhœa, 164.
 Epidermophytides, 402.
 Epilation in ringworm of the scalp, 399.
 Ephedrine hydrochloride in hay-fever, 145.
 in Petit mal, children, 151.
 Prolonged faints, 10.
 Vaso-vagal attacks, 10.
 Epilepsies in childhood, 146.
 Epilepsy, bromides in, 151.
 Bromocarpinæ, syrup in, 151.
 Idiopathic, 151.
 Psychoses after, 79.
 Ketogenic diet in, 151.
 Migraine, 86.
 Sodium biborate in, 151.
 Urethane in, 151.
 Vertigo due to, 101.
 Epileptic fit, 146.
 in meningitis, 149.
 Episcleritis, 313.
 Ergosterol in coeliac disease, 253.
 Irradiated, in hip disease, 298.
 in migraine, 90.
 in phlyctenular conjunctivitis, 319.
 Ergotamine tartrate in migraine, 90.
 Erysipelas, Aolan in, 281.
 Milk injections in, 281.
 Erythema nodosum, aspirin in, 269.
 and acute rheumatism, 266.
 Rheumatism, 133.
 Tubercle, 133, 266.
 Clinical significance of, 265.
 Evipan sodium in biliary colic, 63.
 Eye, Bowman's needle for, 106.
 Diseases, common, of adults, 309.
 of Children, 316.
 Everting upper lid, 103.
 Foreign bodies in, 105.
 Minor surgery of, 103.
 Perforating injuries of the globe, 322.
 Spud, chisel-shaped, 106.
 Niblick, 106.
 Eyelashes, displaced, 107.
 Ingrowing, 310.
 Electrolysis for, 310.
 Exercise in obesity, 27.
 Exogenous obesity, 32.
 Extraction of teeth in pyorrhœa, 166.
 Fainting, 9.
 Fallacies, hay-fever, 141.
 Seasickness, 173.
 Falta, theory of, 28.
 Fango pack, Woodhall Spa, 365.
 Faradism in rheumatoid arthritis, 202.
 Fascial spaces of the palm, 209.
 Fat absorption in coeliac disease, 250.
 Ferri et ammon. cit. in anæmia, 371.
 Coeliac disease, 253.
 Pink disease, 264.
 Ferric chloride test, 2.
 Ferrous sulphate, exsiccated (Glaxo), 371.
 Fibrillation, ventricular, 11.
 Adrenalin in, 12.
 Fibroma, vascular, of the vocal cord, 382.
 Fibrositis, 362.
 Spa treatment for, 303.
 Fingers, lymphangitis of, 206.
 Fistula of the external semi-circular canal, 98.
 Flat foot, 153.
 Acute, 158.
 Adolescent, 155.
 Adult, 157.
 and Gonorrhœa, 158.
 Prostatitis, 158.
 due to short tendo achillis, 154.
 Infantile, 153.
 Spasmodic, 155.
 Flavine dressings in tendon sheath cases, 210.
 Fluorescein in eye troubles, 109, 110.
 Foodstuffs, specific dynamic action of, 27.
 "Force" in coeliac disease, 253.
 Forceps, Graddy's, 107.
 Hoffman's, 335.
 Trotter's, 335.
 Fothergill, colporrhaphy, 115.
 Fractures, ankle-joint, 349.
 Depressed, in children, 17.
 Freckles, 407.
 French's needle in blood transfusion, 67.
 Friedrich's ataxy, 240.
 Frölich's syndrome, 34.
 Frontal sinus, 226.
 Opening, 230.
 Washing out, 226.
 Functional psychosis, 74, 76.
 Fungus infections of the skin, common, 398.
 Furunculosis, 120.
 Bacteriophage in, 281.
 Butyrate of Manganese in, 281.
 Stannoxyl in, 281.
 Vaccines in, 281.
 Way of living in, 280.
 Gall-stones, 59.
 Galvanism in rheumatoid arthritis, 202.
 Gänsslen's extracts, 244.

- Gastric juice injection in pernicious anæmia, 246.
- Gastro-enteritis, acute, 221.
- General paralysis, psychosis of, 79.
- Genital organs and obesity, 35.
- Gerson's diet in hip disease, 298.
- Gigli saws, 334.
- Gingivectomy in pyorrhœa, 165.
- Gingivitis in pyorrhœa, 161.
- Glass probe for wool brush, 105.
- Glaucoma, eserine in, 314.
- Glegg's mixture, in common cold, 170.
- "Glove and stocking" distribution, 241.
- Glucose, hypertonic, in circulatory failure, 10.
in Liver cases, 122.
Pneumonia, 292.
Summer diarrhœa, 212.
- Glucose, intravenous in cardiac failure, 12.
- Glue, Sinclair's, 355.
- Glycerine and phenol, 2 per cent., 195.
- Tannic acid in pyorrhœa, 163.
- Thymol. co., in conjunctivitis, 318.
- Glycerophosphates of iron in phlyctenular conjunctivitis, 319.
- Gold injections in rheumatoid arthritis, 203.
- Gonorrhœa and flat foot, 158.
- Gonorrhœal ophthalmia, Condy's fluid in, 288.
Crédé's method, 317.
Mercurochrome in, 317.
Protargol in, 316.
- Gout, acute and chronic, spa treatment for, 304.
- Gowers on disseminated sclerosis, 53.
- Gowers' Mixture in migraine, 39.
Syndrome, 9.
- Graddy's expression forceps, 107.
- Gravel rash, 324.
- Green line stomatitis, 39.
- Green stools in breast feeding, 213.
Diarrhœa, 222.
- "Grumbling disease" (tubercular hip), 301.
- Gums, massage of, 162.
- Habitual vomiting, 212.
- Hæmatemesis, blood transfusion for, 71.
- Hæmophilia, blood transfusion for, 71.
- Hæmorrhage in electrical injuries, 393.
- Hæmorrhagic disease of the newborn, blood transfusion for, 276.
- Hand, bursæ of, 208.
Infections of the, 205.
Septicæmia from, 207.
- "Hangnail," infected, 206.
- Harrison's course, syphilis, 40.
- Harrogate, bracing Spa, 364.
for rheumatism, 307.
- Hay-fever, ætiology, 139.
Adrenalin in, 145.
"Aklorap" tablets in, 145.
- Asthma, 140.
- Blackley on, 137.
- Bostock on, 137.
- Carbon dioxide gas in, 145.
- Diagnosis, 140.
- Ephedrine hydrochloride in, 145.
- Fallacies, 141.
- Palliatives, 144.
- Perennial, 137.
- Preventives, 142.
- Symptoms, 140.
- Head injuries, in children, 15.
Psychosis after, 78.
Rest after, 19.
- Headache, unilateral, 83.
Periodic, 84.
- Heart-block, 11.
- Heat in rheumatoid arthritis, 201.
- Henoch's purpura, 339.
- Hepatex, 244.
- Hepatic cirrhosis in infants, 49.
- Hernia in children, 345.
- Herpes oticus in children, 193.
Zoster ophthalmicus, 312.
- Herter's infantilism, 248.
- Hexamine in broncho-pneumonia of children, 295.
Disseminated sclerosis, 56.
Jaundice, 124.
- Hip disease, tuberculous, 297.
Abscess in, 301.
Aspiration of, 301.
- Ambulatory methods, 300.
- Beck's plaster, 301.
- Calot's paste, 301.
- Complications, 301.
- Dislocation in, 301.
- Ergosterol, irradiated, for, 298.
- Extra-articular fusion operation, 302.
- Gerson's diet in, 298.
- Jones' abduction frame, 299.
- Local treatment, 298.
- Long plaster spica, 299.
- Parrish's syrup for, 298.
- Pugh's suspension method, 300.
- Sinuses, 301.
- Ultra-violet rays for, 298.
with deformity from muscle spasm, 300.
- Hirschsprung's disease, 339.
- Hoarseness, chronic, 379.

- Hoffman's nibbling forceps, 335.
Hog-spleen injectons for eczema, 329.
 -stomach extract in anæmia, 372.
Hordeolum, 309.
Horner's syndrome in electrical injuries, 396.
Horse serum in migraine, 89.
Horsley's bone-wax, 335.
Hot-air bath, in diabetic coma, 4.
Hydrarg. perchlor. lotion in conjunctivitis, 287.
Hydrochloric acid in hay-fever, 145.
Hydrogen peroxide in pyorrhœa, 163.
"Hygienator" in pyorrhœa, 164.
Hyperinsulism, 28.
Hypertonic salt solution, 16.
Hypoderms in seasickness, 176.
Hypoglycæmia, risk of, 3.
 from insulin, 6.
Hypophyseal obesity, 34.
Hypopyon ulcer, 312.
Hypothyroidism in subacute combined degeneration of the spinal cord, 246.
Hysteria, traumatic, 78.
Icterus gravis neonatorum, 47.
 of the newborn, 46.
Ichthyol in eczema, 329.
Ichthyosis hystrix, 409.
Idiopathic steatorrhœa, 366, 388.
Ileo-colitis, acute, 220.
 Calcium caseinate in, 221.
Impetigo, Bockhart's, 283.
 Contagiosa, 278.
Impletol in migraine, 91.
Infantile convulsions, calciumchloride in, 148.
 Artificial sunlight in, 148.
 Chloral in, 147.
Insanity of climacteric, 76.
Insanity after puberty, 76.
 after traumatic experience, 77.
 sudden, 80, 72.
Insomnia, adalin in, 188.
 Ætiology, 180.
 Allonal in, 188.
 Ammonium bromide in, 185.
 Amylene hydrate in, 188.
 Bromides in, 188.
 Chloral hydrate in, 186, 188.
 Drugs in, 187.
 Factors, physical, 180.
 psychic, 181.
 in children, 184.
 in pleurisy, 128.
 Medinal in, 188.,
 of arterio-sclerosis, 180.
 Paraldehyde in, 188.
 Somnacetin in, 188.
 Sulphonal in, 188.
 Treatment of, 178, 185.
Insulin, coma, 2.
 Omission of, 1.
 Overdose of, 6.
 in rheumatoid arthritis, 203.
 in "summer diarrhœa," 212.
Insurance companies and weight increase, 33.
Interstitial keratitis, 320.
Intestinal indigestion, chronic, 248.
 Infantilism, 248.
 Obstruction, acute, 345.
Intracranial neoplasm, vertigo due to, 111.
Intraperitoneal injection, normal saline, 223.
 Glucose, 223.
 Ringer's solution, 223.
Intravenous injection, sodium chloride, 120.
Intussusception, 338
 Operation for, 346.
Iodide of iron in phlyctenular conjunctivitis, 319.
Iodine in hand infections, 205.
 Pyorrhœa, 163.
 Rheumatoid arthritis, 203.
Iodo-boric powder, 197.
Ionisation in migraine, 91.
 Rheumatoid arthritis, 202.
Ipecacuanha in pyorrhœa, 164.
Irido-cyclitis, 314.
Iritis, 314.
 Diagnosis, 315.
Iron, Sargent's treatment, 245.
 Therapy, 370.
Irradiated ergosterol, 30.
Irwin, Florence, liver recipes by, 243.
Israel (carbuncle kidney), 119.
Jaksch's, von, anæmia, 276.
Jarisch-Herxheimer reaction, 39.
Jaundice, acholuric family, 50.
 Acidosis in, 123.
 Aperients in, 124.
 Appendicitis after, 50.
 Catarrhal, 48
 Digestive disturbance in, 124.
 in childhood, 46.
 in infancy, 46.
 Ketosis in, 123.
 Obstructive, 121.
 Physiological, 46.
 Pruritis in, 123.
 Salicylates in, 124.
Jelopar in common cold, 171.
Jones' abduction frame in hip disease, 299.
"Journal of a Disappointed Man," (disseminated sclerosis) 53.
Joyce, J. L., carbuncle kidney, 119.
Juvenile obesity, 34.

- Kalzana, in jaundice, 123.
 Kaolin, cataplasma, in pleurisy, 127.
 in cholera infantum, 222.
 Kaylene in children's diarrhoea, 293.
 Keratitis, deep, 312.
 Interstitial, 320.
 Superficial, 312.
 Kerion, scalp ringworm, 400.
 Ketonic diet in epilepsy, 151.
 Migraine, 150.
 Ketosis in jaundice, 123.
 Kidney, carbuncle of the, 118.
 Koch-Weeks' bacillus in conjunctivitis, 310.

 Lassar's paste, for eczema, 329.
 Lavis, in pyorrhoea, 164.
 Labyrinthine vertigo, treatment, 98.
 Lachrymal apparatus, affections of, 109, 311.
 Lactic acid milk, in pink disease, 262.
 Laryngitis, chronic, 383.
 Silver nitrate in, 384.
 Tuberculous, 385.
 Laryngo-fissure for cancer, 387.
 Larynx, cancer of, 386.
 radium in, 387.
 Papillomata of, 380.
 Syphilis of, 381.
 Lathyrism, 240, 242.
 Leamington, sedative spa, 364.
 for rheumatism, 307.
 Lee's course, syphilis, 40.
 Leeches, in pleurisy, 127.
 Leg, fractures of, ambulatory treatment, 553.
 Leucoderma, 408.
 Leukæmia, 276.
 Blood transfusion for, 71.
 Lichen urticatus, 329.
 Carbohydrates in, 329.
 Diet in, 330.
 Syr. calc. lactophos. in, 330.
 Lids, inflammatory lesions of, 319.
 Lipiodol radiography, 242.
 Liquid paraffin in nasal catarrh, 228.
 Liver cases, diet in, 123.
 Extract in anæmia, 372.
 Therapy, 373.
 Fish liver, 244.
 in subacute combined degeneration of the cord, 243.
 Intramuscular injection, 377.
 Intravenous therapy, 376.
 of surgical delay, 63.
 Parenteral therapy, 374.
 Recipes by Florence Irwin, 243.
 Lixen in pneumonia of children, 293.
 Llandrindod Wells, bracing spa, 364.
 for rheumatism, 307.
 Llanwetyd Well, sulphur spa, 365.

 London Blood Transfusion Service, 68.
 Lugol's iodine in migraine, 91.
 Lumbago, spa treatment, 304.
 Lumbar puncture, 16.
 in children, 16.
 in electrical injuries, 391.
 Luminal in infantile convulsions, 150.
 Sodium in vertigo, 98.
 Lung, needling, 131.
 Luxus consumption, 27.
 Lymphangitis, fingers, 206.
 Lymphatic leucæmia, 239.
 Lysol in pyorrhoea, 163.

 Magnesium sulphate in concussion, 16.
 in Otitis media, 195.
 Malassez, pityrosporon of, 404.
 Manganese, collosol, in ecthyma, 284.
 Furunculosis, 281.
 Marasmus, blood transfusion in, 71.
 Marie, Pierre, on disseminated sclerosis, 56.
 Marmite in coeliac disease, 253.
 Pink disease, 264.
 Subacute combined degeneration of the spinal cord, 245.
 Massage in disseminated sclerosis, 57.
 Rheumatoid arthritis, 202, 203.
 Subacute combined degeneration of the spinal cord, 247.
 of the gums, 162.
 Mastoiditis in pink disease, 262.
 Mastoidotomy, indications for, 196.
 Meckel's diverticulum, persisting, 338.
 Medinal in insomnia, 185, 188.
 Mediterranean anæmia, 276.
 Melæna, blood transfusion for, 71.
 Melanin, 405.
 Mellanby on disseminated sclerosis, 56.
 Men, obesity in, 32.
 Menière's syndrome, 10, 96.
 Meningitis, epileptic fits in, 149.
 Meningomyelitis, 240.
 Menopausal obesity, 28.
 Mental disorder, acute, 73.
 Cases, treatment, 81.
 Process, factors in, 73.
 Prognosis, 80.
 Treatment, Act of 1930, 81.
 Menthol in common cold, 170.
 Mercurochrome in gonorrhœal ophthalmia, 327.
 Pyorrhoea, 163.
 Mercury in disseminated sclerosis, 56.
 in Syphilis, 39.
 Perchloride compresses, 403.

- Migraine**, 83.
 Abdominal type, 88.
 Allergic type, 89.
 Alimentary type, 88.
 Bile acids in, 92.
 Bilious type, 88.
 Caffeine in, 92.
 Calcium lactate in, 90.
 Cerebral type, 91.
 Collaps, placental hormone, 91.
 Compral in, 92.
 Decholin in, 88.
 Dietetic type, 88.
 Duodenal type, 88.
 Emmemin complex (Glaxo), in, 91.
 Endocrine type, 91.
 Epilepsy, 86.
 Equivalents, 87.
 Fibrositic type, 91.
 Ergotamine tartrate in, 90.
 Ergosterol, irradiated, in, 90.
 Gower's mixture in, 93.
 Horse serum in, 89.
 Ionisation in, 91.
 Ketogenic diet in, 150.
 Lugol's iodine in, 91.
 Major, 85.
 Metabolic type, 89.
 Para-epileptic type, 91.
 Psychological type, 92.
 Ocular type, 90.
 Ophthalmoplegic, 87.
 Peptone in, 89.
 Petit mal in, 150.
 Phenacetin in, 92.
 Psychotherapy in, 92.
 Salicylates in, 91.
 Sinus type, 91.
 Sympathetic type, 90.
 Theelin in, 92.
 Vasomotor type, 90.
 Veramon in, 92.
Migrainous neuralgia, 87.
Milk injections for eczema, 329.
 Erysipelas, 281.
Mongolian spot, 407.
Monilia infections, 404.
Monsol in paronychia, 404.
Morax-Axenfeld conjunctivitis, 310.
Morphia in burns and scalds, 20.
 in Biliary colic, 62.
 Cardiac dyspnoea, 13.
 Coronary thrombosis, 12.
 Dyspnoeic seizure, 14.
 Pleurisy, 128.
Morton-Smart coil in rheumatoid arthritis, 202.
Mouth-washes in pyorrhoea, 164.
Mucous tubercles, 380.
Mud-packs in rheumatoid arthritis, 201.
Myalgia, spa treatment, 303.
Myelitis, 240.
Myocardial infarction, differential diagnosis, 62.
Myringotomy, 195.
N.914 in syphilis, 39.
Nævus unius lateralis, 409.
 Blue, 407.
Nail removal avoided, 206.
 Ringworm of the, 403.
Nasal catarrh, 224.
 Allergic cases, 230.
 and tonsillitis, 228.
 Bicarbonate sodium in, 227.
 Chronic, 228.
 Encephalitis lethargica and, 227.
 Lipiodol X-ray, 229.
 Liquid paraffin in, 228.
 Operation in, 229.
 Polypi in, 231.
 Subacute cases, 227.
 Douches in common cold, 171.
 Inflammation, cocaine in, 226.
Nausea maris, 172.
Needle bath, circular, 358.
Neuralgia, migrainous, 87.
Neurosis, vertigo in, 102.
Newly-born, hæmorrhage in, blood transfusion for, 71.
Night feeds, breast, 213.
Nitritoid crisis in syphilis, 39.
Normal saline, subcutaneously, 234.
Novalgin in rheumatoid arthritis, 201.
Novarsenobillon (N. 914) in disseminated sclerosis, 56.
Novocain in eye cases, 309.
 Infant cases, 234.
Novutox in eye cases, 309.
Obese, mortality rate of, 26.
Obesity, adrenal, 28.
 Constitutional, 28.
 Diet, the Salisbury, 29.
 Salt-free, 30.
 Dietary treatment, 30.
 Endocrine, 28.
 Endogenous, 27, 32, 34.
 Exercise in, 27.
 Exogenous, 27, 32.
 Genital organs and, 35.
 Hypophyseal, 34.
 Juvenile, 34.
 Men, 32.
 Diet treatment, 36, 37.
Menopausal, 28.
Psychological factor, 30.
Pituitary, 28.

- Obesity :
 Thyroid, 34.
 in female, 28.
 Treatment of, 29.
 Types of, 27.
 Women, 26.
 Yeast extracts in, 30.
- Ophthalmia neonatorum, 288.
- Opium in burns and scalds, 20.
 in Diarrhœa, 220.
 Otitis media, 195.
- Orange juice in anæmia of infancy, 273.
- Orris hay-fever, 142.
- Orris-root-free cosmetics, Boutall's, 142.
- Osteo-arthritis, spa treatment for, 304.
- Otitis media in children, 189.
 Benign, 192.
 Differential diagnosis, 193.
 Hæmolytic, 191.
 Histiolytic, 192.
 Magnesium sulphate in, 195.
 Mucous, 192.
 Pneumococcal, 192.
 Prophylaxis, 190.
 Purulent, 192.
 Toxic, 191.
 Treatment, 194.
 Types of, 191.
 Warnings, 195.
- Out-door treatment of pneumonia, 291.
- Ovarian extract in rheumatoid arthritis, 203.
- Ovaries in female obesity, 28.
- Oxycyanide of mercury in conjunctivitis, 310.
- Oxygen apparatus, Davies and Gilchrist, 292.
 in Coronary thrombosis, 12.
 Broncho-pneumonia of children, 295.
- Palm, collar-stud abscess in, 208.
 Fascial spaces of the, 209.
- Palpitation, 13.
- Pancreatitis, acute, differential diagnosis, 62.
- Pannus after trachoma, 289.
- Papillomata of larynx, 380.
- Paracentesis thoracis, 129.
- Paralysis of the vocal cords, 388.
- Parenchymatous keratitis, 320.
- Paraffin, liquid, for antrum lavage, 226.
- Paraldehyde in insomnia, 188.
- Paronychia, 206.
 Monsol in, 404.
- Paroxysmal tachycardia, 13.
 Syncope in, 11.
- Parrish's syrup in hip disease, 298.
- Parke, Davis' spray in common cold, 171.
- Peat baths, Harrogate, 364.
 Strathpeffer, 365.
- Pediculosis capitis, carbolic lotion for, 325.
 Corporis. lot. hydrarg. perchlor. in, 279.
 Sulphur in, 324.
- Pubis, on eyelashes, 325.
- Pellagra, 240, 242.
- Pelvic floor prolapse, 112.
- Peptic ulcer, blood transfusion for, 71.
- Perineum, torn, repair of, 114.
- Peripheral vascular failure, 8.
- Peritonitis, pneumococcal, 338.
 Streptococcal, 338.
 Operation in, 346.
 Tuberculous, diagnosis, 251.
- Permanganate of potash in colon lavage, 360.
 in Rheumatoid arthritis, 203.
- Pernicious anæmia, Castle on, 245.
 Gastric juice injections in, 246.
 Strauss on, 245.
 Subacute combined degeneration of cord in, 373.
- Perthe's disease, diagnosis, 297.
- Pessary treatment, 113.
- Petit mal, in children, ephedrine for, 151.
 in Pyknolepsy, 150.
 Syphilis, 150.
- Phlyctenular conjunctivitis, atropine in, 319.
- Pigment formation in the skin, 405.
- Pink disease, adexolin in, 264.
 Adrenalin in, 264.
 Almata in, 261.
 Arsenic in, 259, 264.
 Calcium lactate in, 261, 264.
 Ferri et ammon. cit. in, 264.
 Lactic acid milk in, 262.
 Marmite in, 264.
 Mastoiditis in, 262.
 Pituitrin in, 264.
 Radiomalt in, 263.
 Radiostol in, 259, 264.
 Signs, 256.
 Strychnine in, 264.
 Therapeutics, 263.
 Trichotillomania in, 257.
 Ultra-violet light in, 259, 263.
- Pink eye in children, silver nitrate in, 317.
- Pinoleum in common cold, 171.
- Pituitrin in diabetic coma, 6.
- Plasticine, fibrous, ear-plug, 198.
- Pleural effusions and rheumatism, 133.
 Transudates, 133.
- Shock, 131.

- Pleurisy, acute dry, 126.
 Dry cupping in, 127.
 Leeches in, 127.
 with effusion, 128.
 Aspiration in, 129.
 Aspirin in, 128.
 Cataplasma kaolin in, 127.
 Children, caloplast for, 294.
 Dover's powder in, 128.
 Dry chronic, 128.
 Cupping in, 127, 129.
 Leeches in, 127.
 Morphia in, 128.
 Siphonage in, 129.
 Treatment of, 124.
 Veramon in, 128.
- Pleurodynia, spa treatment for, 303.
- Pneumonia after common cold, 171.
 Differential diagnosis, 62.
 Glucose in, 292.
 in children, 291.
- Pollen dermatitis, 140.
 Solutions (Duncan Flockhart), 142.
- Pollens for testing (Duncan Flockhart), 142.
 in hay-fever, 137.
- Polypi, aural, 196.
 in nasal catarrh, 231.
- Port wine on infant's comforter, 234.
- Potassium bismuth tartrate in syphilis, 39.
 Iodine in disseminated sclerosis, 56.
 Syphilis, 40.
- Pott's fracture, 349.
- Pottolites in rheumatoid arthritis, 201.
 for arm, 207.
- Proetz, displacement method of, in nasal catarrh, 229.
- Prolapse of the uterus, 112.
- Prostatitis and flat-foot, 158.
- Protargol in conjunctivitis, 287.
- Protargol in gonorrhoeal ophthalmia, 316.
- Protein milk in coeliac disease, 252.
- Protosol in ileo-colitis, 221.
- Pruritis, alkaline baths for, 124.
 in jaundice, 123.
 Thyroid in, 123.
- Psychoses, acute, 73, 77.
 Endocrine imbalance in, 77.
 Functional, 74, 76.
 of general paralysis, 79.
 Organic, 74, 78.
 Symptoms, 75.
 Reactive, 75.
 Reductive, 75.
 Release, 75.
- Psychotherapy in migraine, 92.
- Pugh's method of suspension, hip disease, 300.
- Pulmonary embolism, 13.
 Edema, acute, 13.
- Purpura, blood transfusion for, 71.
- Pyelitis, 339.
- Pyelography, 119.
- Pyloromyotomy, 234.
- Pylorospasm, 212.
- Pyorrhoea alveolaris, 159.
 Ammonium chloride in, 165.
 Arsenic in, 163, 165.
 Brodrick, F. W., on, 165.
 Cautery in, 165.
 Chromic acid in, 163.
 Copper sulphate in, 164.
 Creosote and alcohol in, 163.
 Curettage in, 165.
 Drug treatment, 163.
 Electro-therapy in, 166.
 Emetine in, 164.
 Endocrine disturbance in, 161.
 Entamoeba buccalis in, 164.
 Extraction of teeth in, 166.
 General treatment, 165.
 Gingivectomy in, 165.
 Gingivitis and, 161.
 Glycerine and tannic acid in, 163.
 Hydrogen peroxide in, 163, 164.
 "Hygienator" in, 164.
 Iodine in, 163.
 Ipecacuanha in, 164.
 Lysol in, 163.
 Mechanical treatment, 162.
 Mercurochrome in, 163.
 Mouth-washes in, 164.
 Surgical treatment, 164.
 Vaccines in, 166.
 Zinc chloride in, 164.
- Pyramidon in erythema nodosum, 270.
 Rheumatoid arthritis, 201.
- Queckenstedt's test, 242.
- Quinidine in tachycardia, 14.
 Intravenous, 14.
- Quinine in disseminated sclerosis, 56.
 Vertigo, 99.
- Radiostoleum in infantile convulsions, 148.
 in Subacute combined degeneration of the spinal cord, 245.
- Radium burns, tannic acid in, 25.
 In Cancer of the larynx, 387.
- Rammstedt's operation, 212, 234.
- Rectal examination in a child, 341.
 Saline after Rammstedt's operation, 237.
- Suppositories in seasickness, 177.
- Rectocele, 112, 113.

- Rheumatism, Bath Spa for, 307.
 Droitwich Spa for, 307.
 Leamington Spa for, 307.
 Llandrindod Wells, 307.
 Strathpeffer Spa for, 307.
 Trefriw Wells Spa for, 307.
- Rheumatoid arthritis, 199.
 Antiphlogistine in, 201.
 Aspirin in, 201.
 Atophan group in, 201.
 Blood transfusion in, 203.
 Caffeine in, 201.
 Codeine phosphate in, 201.
 Diathermy in, 202.
 Diet in, 203.
 Electric pads in, 201.
 Faradism in, 202.
 Galvanism in, 202.
 Gold injections in, 203.
 Insulin in, 203.
 Iodine in, 203.
 Ionisation in, 202.
 Morton Smart coil in, 202.
 Mud-packs in, 201.
 Novalgin in, 201.
 Poultices in, 201.
 Prognosis, 204.
 Pyramidon in, 201.
 Skin in, 203.
 Sodium salicylate in, 201.
 Spa treatment in, 304.
 Thyroid in, 203.
 Ultra-violet light in, 203.
 Vaccines in, 203.
- Rickets, coeliac, 249.
 Black dot, 399.
 Kerion, 400.
- Ringworm of the beard, 403.
 Glabrous skin, 326, 401.
 Scalp, 327, 398.
 Thallium in, 327, 400.
 Whitfield on, 400.
 X-ray in, 327, 400..
 Toes, 402.
- Roger's spray, 226.
 Rothera's nitro-prusside test, 2.
 Rubber tissue wicks for drainage, 210.
 Rumination, 212.
 "Runaround," 206.
- Salicylates in jaundice, 124.
 "Salmon patch," 320.
 Sargant's iron treatment, 245.
 Scabies, ecthyma after, 284.
 Sulphur dermatitis, cyllin for, 326.
 Scalds, mortality from, 24.
 Treatment of, 20.
 Scaling of teeth, 162.
 Sciatica, spa treatment for, 304.
- Sclerotic, affections of, 313.
 Scrum pox, 324.
 Seasickness, 172.
 Atropine in, 176.
 Chloral hydrate in, 177.
 Chloretone in, 177.
 Fallacies, 173.
 Liq. trinitrini in, 176.
 Luminal in, 99.
 Pot. bromid. in, 173, 177.
 Sod. bicarb. in, 174.
 Strychnine in, 176.
 Treatment of, 172.
 Valerianates in, 177.
 Vertigo in, 99.
- Semon's law in laryngeal paralysis, 388.
 Septicæmia from hand, 207.
 Serum-resistant cases of syphilis, 44.
 Shock, in diabetic cases, 4.
 Silver nitrate in conjunctivitis, 310.
 in Chronic laryngitis, 384.
 Trachoma, 288.
- Silversalvarsan in disseminated sclerosis, 56.
 Sinclair's glue, 355.
 Singers' nodules, 383.
 Sinusitis, 225.
 Ethmoidal opening, 230.
 in children, 189.
 Sphenoidal, 225.
 Opening, 230.
- Sitz-bath, aerated, 356.
 Skin diseases, common, of children, 323.
 Due to cocci, 278.
- Skin, fungus infections of the, 398.
 Skull, basal fractures of, 18.
 Compound fractures of, 17.
 Depressed fractures of, 17.
 Plough for osteoplastic craniotomy, 335.
- Sleep centre, 179.
 Nature of, 178.
 Partial, 179.
 Theories concerning, 178.
- Sleeplessness, effects of, 179.
 Snuffles, 380.
- Sodium biborate in epilepsy, 151.
 Bicarbonate in diabetic coma, 4.
 Chloride, intravenous, 16.
- Somnacetin in insomnia, 188.
- Spa facilities, a practical survey of, 356.
 Treatment, definition of, 304.
 Brachial neuritis, 304.
 Chronic rheumatism, 307.
 Torticollis, 303.
 Waters, chemical constituents of, 305.
- Spas, individual, 362.
 Sparklet, carbon dioxide from, 145.

- Spasmophilia, 148.
 Spastic paraplegia (?), 240.
 Spinal cord, subacute combined degeneration of, 239.
 a deficiency disease, 245.
 Adexolin in, 245.
 Avolesum in, 245.
 Bedsore in, 246.
 Bemax in, 245.
 Dimol in, 247.
 Liver in, 243.
 Marmite in, 245.
 Radiostoleum in, 245.
 Yeast in, 245.
 Splenic anæmia, 49.
 Splint, Delbet, for ankle-joint, 353.
 Sprays in common cold, 170.
 Spa, 358
 Sprue, 366.
 Non-tropical, 248.
 Squint, 321.
 Stabilarsan, 41.
 Stannoxyl in furunculosis, 281.
 Status epilepticus, 152.
 Chloral injections in, 152.
 Chloroform inhalation in, 152.
 Hyoscin hydrobromide in, 152.
 Paraldehyde in, 152.
 Status hemicanicus, 86.
 Stomach wash-out, 222.
 Stomatitis, "green line," 39.
 Stones, H., on gingivectomy, 165.
 Strathpeffer sulphur spa, 365.
 Strophanthin, in acute cardiac dyspnoea, 13.
 Intravenously, 14.
 Strychnine in cholera infantum, 223.
 in Peripheral failure, 10.
 Respiratory distress, 296.
 Syncope, 14.
 Styte, 108.
 Sulpharsenol in syphilis, 42.
 Sulphonal in insomnia, 188.
 Sulphosin Leo in disseminated sclerosis, 55.
 Sulphostab in syphilis, 42.
 Summer diarrhoea, 212.
 Normal saline in, 212.
 Sunburn, tannic acid in, 25.
 S.U.P. 36 (B.D.H.), in influenza, children, 295.
 Broncho-pneumonia, children, 295.
 S. 606 in syphilis, 39.
 Sycosis, coccogenic, 282.
 Butyrate of manganese in, 283.
 Syncope, adrenalin in, 14.
 Syphilis, arsenic in, 38, 40.
 Arterio-sclerosis in, 39.
 Bismuth in, 39.
 Burke's method, 41.
 Clinical relapse, 44.
 Syphilis:
 Congenital, 48, 379.
 Drake and Thomson's course, 41.
 Dermatitis in, 39.
 Harrison's course, 40.
 Larynx, 384.
 Lee's course, 40.
 Mercury in, 39.
 N. 914 in, 39.
 Nitritoid crisis in, 39.
 Potassium bismuth tartrate in, 39.
 iodide in, 40.
 Psychological factor, 43.
 S. 606 in, 30.
 Serum-resistant cases, 44.
 Sulpharsenol in, 42.
 Sulphostab in, 42.
 Thiobismol in, 39.
 Treatment, 38.
 Whitfield's course, 41.
 Syrup ferri iodidi in phlyctenular conjunctivitis, 289.
 Tachycardia, intravenous, quinidine in, 14.
 Paroxysmal, 13.
 Tannic acid, dressing for burns, 24, 20.
 Tablets, 20.
 Teachers' nodes, 383.
 Teichopsia, 84.
 Teeth, septic, psychosis after extraction, 80.
 Tendon sheath infections, 208.
 Tenosynovitis lesions, 209.
 Test, Benedict, 2.
 Thermogene wool in pleurisy, 127.
 Thyroid in infantile convulsions, 150.
 Pruritis, 123.
 Tinea circinata, 401.
 Cruris, 402.
 Versicolor, 403.
 Toe, ringworm of, 403.
 Tonsillitis and nasal catarrh, 228.
 Toxic conditions, psychoses of, 79.
 Trachoma, 289.
 Copper sulphate in, 289.
 Silver nitrate in, 289.
 Transillumination, nasal, 225.
 Trefriw, sedative tonic spa, 365.
 Trichophytides, 402.
 Tuberculous hip-disease, 297.
 Peritonitis, diagnosis, 251.
 Transfusion, blood, *see* Blood transfusion.
 Ultra-violet light, blue filter on, 399.
 in Children's convalescence, 295.
 Ecthyma, 284.

- Ultra-violet light :
 in Hip disease, 298.
 Rheumatoid arthritis, 203.
 Umbilicus, infection of the, 48.
 Undine, drop bottle, 104.
 in conjunctivitis, 287.
 Ungley's raw brain treatment, 244.
 Ung. hyd. ox. flav. in phlyctenular
 conjunctivitis, 289.
 Urethane in epilepsy, 151.
 Urinary tract, coccal infections of
 the, 118.
 Urotropin after skull operations, 18.

 Vaccine therapy in common cold,
 169.
 in Furunculosis, 281.
 Pyorrhœa, 166.
 Rheumatoid arthritis, 203.
 Van den Bergh reaction, 47, 48.
 in biliary colic, 61.
 Vascular fibroma of the vocal cord,
 382.
 Vaso-vagal attacks, 9.
 Venesection in cardiac asthma, 13.
 Pulmonary œdema, 13.
 Ventricular failure, 11.
 Ventriculin, 244.
 Ventrofixation, 114.
 Vertigo, aural, 97.
 Acute labyrinthine, 97.
 due to cerebral contusion, 101.
 Circulatory disturbances, 101.
 Defective postural stability, 100.
 Fistula of the external semi-
 circular canal, 100.
 Disseminated sclerosis, 101.
 Epilepsy, 101.
 Intracranial neoplasm, 101.

 Vertigo :
 in Neurosis, 102.
 Seasickness, 99.
 Labyrinthine, 98, 96.
 Lesions of the vestibular nerve,
 100.
 Luminal sodium in, 98.
 of Central origin, 100.
 of Ocular origin, 96.
 Quinine in, 99.
 Recurrent, 97.
 The nature of, 94.
 Vichy methods of muscle massage,
 305.
 Vitamin content, 30.
 Vitiligo, 408.
 Vocal cords, paralysis of, 388.
 Voice, breaking of, at puberty, 381.
 Vomiting, habitual, 212.

 Wagstaffe's fracture, 350.
 Whitfield's ointment, 400, 403.
 Williamson on disseminated sclerosis,
 56.
 Woodhall Spa, bracing, 365.
 for rheumatism, 307.

 X-ray baldness, 327.
 Burns, tannic acid in, 25.
 Examination of skull in chil-
 dren's injuries, 18.
 in ringworm of the scalp, 327,
 400.
 Xanthochromia in spinal fluid, 242.

 Zinc chloride in blepharitis, 319.
 in Conjunctivitis, 287, 289, 311.
 Pyorrhœa, 164.

—LEWIS'S GENERAL PRACTICE SERIES—

RHEUMATISM IN GENERAL PRACTICE. A Clinical Study.

By MATTHEW B. RAY, M.D.(Edin.). With a Foreword by LORD HORDER, K.C.V.O., M.D., F.R.C.P. With 6 Plates. Demy 8vo. **16s.** net

COMMON SKIN DISEASES.

By A. C. ROXBURGH, M.D., F.R.C.P. With 8 Coloured Plates and 110 Illustrations in the Text. Demy 8vo. **18s.** net.

CHEST DISEASE IN GENERAL PRACTICE, with Special

Reference to Pulmonary Tuberculosis. By PHILIP ELLMAN, M.D., M.R.C.P. With a Foreword by PROF. S. LYLE CUMMINS, C.B., C.M.G., M.D. With 132 Illustrations. Demy 8vo. **15s.** net

AFFECTIONS OF THE EYE IN GENERAL PRACTICE.

By F. LINDSAY REA, M.D., F.R.C.S., With 7 Coloured Plates and 35 other Illustrations. Demy 8vo. **10s. 6d.** net.

THE CONDUCT OF LIFE ASSURANCE EXAMINATIONS.

By E. M. BROCKBANK, M.D. (Vict.), F.R.C.P. Demy 8vo. **7s. 6d.** net.

OTHER VOLUMES IN PREPARATION.

A SHORT PRACTICE OF SURGERY.

By HAMILTON BAILEY, F.R.C.S. (Eng.), and McNEILL LOVE, M.S., (Lond.), F.R.C.S. (Eng.). In Two Volumes. With 618 Illustrations. Demy 8vo. **20s.** net, each

SURGICAL ANATOMY AND PHYSIOLOGY.

By NORMAN C. LAKE, M.D., M.S., D.Sc., F.R.C.S., and C. JENNINGS MARSHALL, M.D., M.S., F.R.C.S. 238 Illustrations. (Many Coloured) Demy 8vo. **30s.** net. *Just Published.*

RECENT PROGRESS IN MEDICINE AND SURGERY, 1919-1933.

Edited by SIR JOHN COLLIE, C.M.G., M.D. In One Volume. Demy 8vo. With 38 Illustrations. With a Foreword by LORD HORDER. **16s.** net

THE PRINCIPLES AND PRACTICE OF RECTAL SURGERY.

By W. B. GABRIEL, M.S. (Lond.), F.R.C.S. (Eng.). With 8 Coloured Plates and 110 Illustrations. Royal 8vo. **20s.** net.

THE PRINCIPLES AND PRACTICE OF OTOTOLOGY

By F. W. WATKYN-THOMAS, F.R.C.S., B.Ch.(Camb.), and A. LOWNDEN YATES, M.C., M.D., F.R.C.S. With 199 Illustrations. Demy 8vo. **25s.** net

Lewis's Publications are obtainable at all booksellers.

London : H. K. LEWIS & Co. Ltd., 136 Gower Street, W.C.1

THE MEDICAL PRESS AND CIRCULAR

1200 DOCTORS

have become subscribers to the "MEDICAL PRESS AND CIRCULAR" in less than eighteen months. There could hardly be a stronger endorsement of the policy adopted by the Editors. That policy is stated on the opposite page.

EDITORIAL STAFF

CECIL P G WAKELEY,
D Sc F.R.C.S. F.R.S.E.

M. SYDNEY THOMSON
M.A. M.D., F.R.C.P.

H. AUDLEY LUCAS, B.A., M.R.C.S. L.R.C.P

EDITORIAL POLICY

IT IS THE POLICY of the Editors of "THE MEDICAL PRESS AND CIRCULAR" to put before the profession, concisely and authoritatively, the latest knowledge in the form in which it can be applied in General Practice. Special features of the Journal are:—

1. **Quarterly Symposiums.** 24 page illustrated Supplements on such subjects as Medical Emergencies (Jan.), Road Accidents (Apr.), Allergic Diseases (July), etc
2. **Special Numbers** Six times a year a whole issue of the Journal is devoted to one subject, *e.g.*, "Maladies of Middle Age" (June).
3. **Clinical Abstracts.** Selected from 40 of the principal journals of the world. The abstracts aim at being practical
4. **Clinical Cases.** Illustrated cases are published frequently.

SOME CONTRIBUTORS:

SIR HUMPHRY ROLLISTON, B1
K.C.B., G.C.V.O., M.D.
SIR ST. CLAIR THOMSON, M.D.,
F.R.C.P., F.R.C.S.
PROFESSOR D. P. D. WILKIE, M.Ch.,
F.R.C.S.
SIR G. LESTER CHEATLE, K.C.B.,
C.V.O., F.R.C.S., F.A.C.S.
SIR HAROLD GILLIES, C.B.E., F.R.C.S.
SIR CHARLOTTE BRISCOE, B1, M.D.
F. CHIWODE CRAWLEY, M.D.
SIR D'ARCY POWER, K.B.E., F.R.C.S.

SIR ARTHUR KEITH D.Sc., F.R.C.S.,
M.D., I.L.D.
SIR BUCKSTON BROWN, F.R.C.S.
SIR HENRY H. GAUVAIN, M.D.,
F.R.C.S.
SIR JOHN COLLIE, C.M.G., M.D.
E. MAPOTHEE, M.D., F.R.C.P.,
F.R.C.S.
W. RUSSELL BRAIN, D.M., M.R.C.P.
L. S. T. BURFELL, M.D., F.R.C.P.
PROFESSOR J. A. GUNN, D.Sc., M.D.
E. G. SLESINGFR, O.B.E., F.R.C.S.



TRIAL SUBSCRIPTION

For **10s. 6d.** (half the annual subscription) the Journal *with Quarterly Symposiums* will be mailed for NINE months. You can thus judge its value for yourself.

8, HENRIETTA STREET, LONDON, W.C.2

BUTTERWORTH & CO. (INDIA) LTD.
CALCUTTA • BOMBAY • MADRAS

Large supplies of
ALL THE LEADING
MEDICAL BOOKS
always in stock



SOLE AGENTS for many important
 works. Write for any books required

CATALOGUES supplied on application.
 Communications to nearest branch

INDIA : BUTTERWORTH & CO. (INDIA) LTD.
CALCUTTA - - 6, Hastings Street (P.O. Box 251)
BOMBAY - Jehangir Wadia Bldg., Esplanade Road
MADRAS - - - 317, Linga Chetty Street



MANY EDITIONS

In the world of books there is no surer guide to the value of a work than the frequency with which new and revised editions are required. The following publications have all proved their worth in this way. The current editions will in every case be found to be an improvement upon all earlier ones.

<i>Editions</i>	<i>Title and Author</i>	<i>Price</i>
14	ROSE & CARLESS' Surgery	30s
12	WHITLA'S Pharmacy	12s. 6d
12	STEDMAN'S Medical Dictionary	35s
10	PARK & WILLIAMS' Pathogenic Micro-organisms	35s
10	CABOT'S Physical Diagnosis	22s 6d
8	BAILEY'S Histology	25s
7	WHITLA'S Dictionary of Treatment	25s
7	CHAPIN & ROYSTER'S Pædiatrics	35s
6	LANGDON BROWN'S Physiological Principles	10s 6d
6	WILLIAMS' Minor Maladies	10s. 6d
6	WORTH'S Squint	10s 6d
6	MAY & WORTH'S Diseases of the Eye	15s
6	KANAVEL'S Infections of the Ear	35s
5	LAMB'S Throat, Nose and Ear	12s 6d
5	MUMMERY'S After-Treatment	7s 6d.
5	TREDGOLD'S Mental Deficiency	25s
5	LAKE & PETER'S Diseases of the Ear	12s. 6d
5	MATHEWS' Physiological Chemistry	35s.
5	BUCHANAN'S Anatomy	35s
5	CLARKE'S Refraction	8s 6d.

In the 'Students' Aids' Series the following volumes have also been revised on several occasions.

10	OBSTETRICS,	by Leslie Williams	3s 6d.
9	POCKET ANATOMY,	by C. H Fagge	5s
8	GYNÆCOLOGY,	by Prof. R. E Tottenham	3s 6d.
7	OPHTHALMOLOGY,	by Bishop Harman	3s. 6d
6	DISEASES OF CHILDREN,	by J. McCaw	4s. 6d.
6	BACTERIOLOGY,	by W. Partridge	5s.
6	PATHOLOGY,	by Harry Campbell	4s. 6d.
5	SURGERY,	by J. Cunning	4s. 6d.

4 Editions, 5 Titles.

3 Editions, 5 Titles.

Baillière Tindall
and Cox. Est. 1826

7 & 8, Henrietta Street, London, W.C.2.

The Prescriber

A Review of the Progress of Medical Science

FOUNDED IN 1906

All the latest developments of Medical Science, particularly with regard to Treatment, are reviewed each month, the literature of the whole world being brought into requisition

THE MOST USEFUL REFERENCE-JOURNAL
FOR THE GENERAL PRACTITIONER

SPECIAL NUMBERS

are issued at intervals. These deal with such subjects as

ANÆMIA

SKIN DISEASES

ANÆSTHETICS

SPA TREATMENT

DIABETES

ENDOCRINOLOGY

Each of these numbers contains a lengthy Review embodying a year's work on the subject. These Reviews are a much appreciated feature of this journal, as they present all the latest developments in a form not obtainable elsewhere.

Published on the first day of each month.

SUBSCRIPTION, 20 shillings per annum,
post free in British Empire.

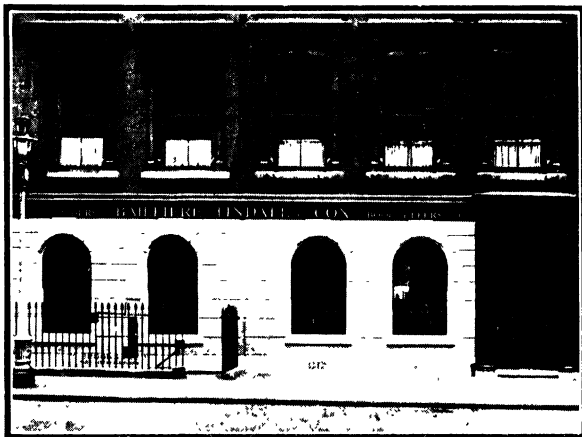
Specimen Copy Free on application.

Obtainable through any Bookseller or Newsagent
or direct from

"THE PRESCRIBER" OFFICES, 13 Glencairn Crescent, EDINBURGH 12

BAILLIÈRE, TINDALL & COX

Medical Publishers and Booksellers



DEPARTMENTS :

Publishing

Sales

Periodical

The Sales Department carries a full stock of all current medical books and books of the allied sciences, both English and American. Any book can be promptly supplied.

The Periodical Department arranges for subscriptions to all English and Foreign Journals.

Both Departments specialize in supplying Libraries, Government Departments Institutions, etc.,

*Write for any
information you require*



*When in London
visit our showrooms*

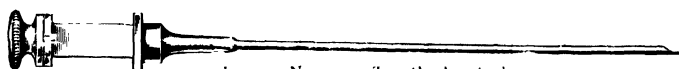
7 & 8, HENRIETTA STREET, COVENT GARDEN, LONDON, W.C. 2.

Always sharp— always clean

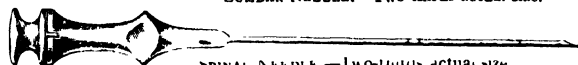
—with interiors as “free” and rustless as their bright, polished exteriors, SOLILA Stainless Needles will always serve you faithfully and well. From their trouble-free unbreakable heads to their keen lancet-like points, Solila Needles are dependably safe and efficient.

Made from Firth-Brearley Stainless Steel, they maintain their sharpness and serviceability for a much longer period than ordinary steel needles and thus are the most economical needles you can buy.

The SOLILA Surgical Needles illustrated represent a few of the popular patterns always in stock. Special patterns can be made to order, in minimum quantities of one dozen, on receipt of a specimen. Send for Free Sample Needle stating pattern required (Lumbar, Spinal and Filling Needles excluded).



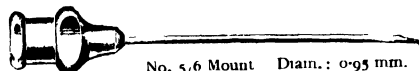
LUMBAR NEEDLE.—Two-thirds actual size.



SPINAL NEEDLE.—Two-thirds actual size



FILLING NEEDLE.—5/6 Mount — Actual size



No. 5/6 Mount Diam.: 0.95 mm. Length: 42 mm

SOLILA STAINLESS STEEL SURGICAL NEEDLES

WARNING.—For the manufacture of Hypodermic Needles in Great Britain, the right of using Stainless Steel of the composition defined in the Brearley patent supplied by steel makers licensed by the Firth-Brearley Stainless Steel Syndicate, Ltd., is reserved to THE AMALGAMATED DENTAL COMPANY, LTD., LONDON, and their Nominees

SOLE WHOLESALE DISTRIBUTORS:

THE AMALGAMATED DENTAL CO., LTD., 7, SWALLOW ST., PICCADILLY, LONDON, W.1



DOUGLAS · J · KIDD

MEDICAL ARTIST

CONTRIBUTORS TO THE MEDICAL PRESS HAVE AT THEIR DISPOSAL THE SERVICES OF AN ILLUSTRATOR WHOSE ACKNOWLEDGED ARTISTIC ABILITY IS BACKED BY FIFTEEN YEARS OF EXPERIENCE IN THE ILLUSTRATION OF SURGICAL, MEDICAL AND SCIENTIFIC LITERATURE.

YOUR ENQUIRIES WILL BE ASSURED OF IMMEDIATE AND EXPERT ATTENTION.

ILLUSTRATIONS IN
COLOUR, LINE OR
MONOCHROME
TERMS ON APPLICATION

Address Your Enquiries
DOUGLAS · J · KIDD,
"BEVERLEY"
ALMONDS GREEN
LIVERPOOL 12

Telephone: Old Swan 137

*Some Recent Issues:***TEXT-BOOK OF THE PRACTICE OF MEDICINE**

By Various Authors, edited by FREDERICK W. PRICE,
M.D., F.R.S.Edin. 4th ed. Pp. 2,040. 106 Illus. 36s. net.

APPLIED PHYSIOLOGY

By SAMSON WRIGHT, M.D., F.R.C.P. 5th ed. Pp. 636.
195 Illus. 18s. net.

DISEASES OF THE NERVOUS SYSTEM

By W. RUSSELL BRAIN, M.A., D.M., F.R.C.P. Pp. 915.
51 Illus. 27s. 6d. net.

OPERATIVE SURGERY

By ALEXANDER MILES, M.D., LL.D., F.R.C.S.; and
D. P. D. WILKIE, M.D., F.R.C.S. Pp. 608. 321 Illus. 21s. net.

THE ENLARGED PROSTATE AND PROSTATIC OBSTRUCTION

By KENNETH M. WALKER, F.R.C.S., M.A., M.B., B.C.
2nd ed. Pp. 236. 68 Illus. (6 in Colour). 14s. net.

THE DIET BOOK, for Doctor, Patient and Housewife

By MARGUERITE REQUA REA, with Foreword by
SIR JAMES PURVES-STEWART. 2nd ed. Pp. 224. 6s. net.

PARALYSIS IN CHILDREN

By R. G. GORDON, M.D., D.Sc., F.R.C.P., and M.
FORRESTER-BROWN, M.D., M.S. Pp. 336. 116 Illus.
15s. net.

THE ANÆMIAS

By JANET M. VAUGHAN, D.M., M.R.C.P., with Notes by
HUBERT M. TURNBULL, D.M., F.R.C.P. Pp. 260.
24 Illus. 12s. 6d. net.

NEPHRITIS AND ALLIED DISEASES

By ROBERT PLATT, M.D., M.R.C.P. Pp. 176. 8 Illus.
7s. 6d. net.

ESSENTIALS OF INFANT FEEDING AND PÆDIATRIC PRACTICE

By HENRY P. WRIGHT, M.D. Pp. 222. 12s. 6d. net.

MAYOU'S DISEASES OF THE EYE

Revised and largely re-written by FREDERICK RIDLEY,
B.Sc., M.B., B.S., F.R.C.S., and ARNOLD SORSBY, M.D.,
F.R.C.S. 4th ed. Pp. 270. 38 Figures. 6s. net.

INJURIES AND SPORT

By C. B. HEALD, C.B.E., M.D. Pp. 567. 380 Illus. 25s. net.

COMPLETE CATALOGUE OF MEDICAL BOOKS FREE ON REQUEST.

Oxford University Press

HUMPHREY MILFORD AMEN HOUSE LONDON, E.C.4



MEDICAL TREATMENT SOME CASSELL BOOKS

SICK CHILDREN :

DIAGNOSIS AND TREATMENT

By DONALD PATERSON, B.A. Manitoba., M.D. Edin.,
F.R.C.P. Lond.

Crown 8vo, 542 pages. With 16 Half-tone Plates
and 85 Text-figures. 16s. net.

MODERN MEDICAL TREATMENT

By E. BELLINGHAM-SMITH, M.D., F.R.C.P. Lond.,
and ANTHONY FEILING, M.D., F.R.C.P. Lond.

Two Volumes. Demy 8vo, 1,432 pages. 30s. net
the set.

PULMONARY TUBERCULOSIS

MEDICAL & SURGICAL TREATMENT

By H. MORRISTON DAVIES, M.A., M.D.,
M.Ch. Cantab., F.R.C.S. Eng.

Demy 8vo, 464 pages. With 77 Radiographic Plates
and 69 Text-figures. 27s. 6d. net.

INJECTION TREATMENT IN

MEDICAL TREATMENT

By DAVID LEVI, M.B., M.S., F.R.C.S. Eng.

Crown 8vo, 158 pages. With 26 Illustrations. 6s. net.

MATERIA MEDICA AND THERAPEUTICS

By W. J. DILLING, M.B., Ch.B. Aberd.

Fourteenth Edition. Entirely Revised. Foolscap
8vo, 700 pages. 10s. 6d. net.

THE TREATMENT OF EPILEPSY

By FRITZ B. TALBOT, M.D.

Demy 8vo, 322 pages. With 11 figures and 29 Tables.
18s. net.

Complete List of Medical Books, post free, on application.

Cassell & Co. Ltd., La Belle Sauvage, London, E.C.4

The Relief of Pain in Childbirth

By F. NEON REYNOLDS, M.C.O.G., F.R.C.S.Ed.

**A concise but detailed account of modern
methods for the relief of pain in childbirth.**

- | | |
|--|--|
| CHAPTER I Historical and Introductory
First Use of Anæsthetics in Midwifery | CHAPTER IV. Drugs and Methods available
and suitable for the First Stage |
| CHAPTER II Preliminary Details of Great
Importance The Conduct of the
Months of Pregnancy The Treatment of
the more Common Discomforts, Mental
and Physical, associated with Pregnancy | CHAPTER V Drugs and Methods available
and suitable for the Second Stage. |
| CHAPTER III General Considerations The
Differing Requirements of the Two
Stages of Labour The Properties Re-
quired in the Drugs and Methods to be
used | CHAPTER VI The Midwife. The Position
regarding Patients attended only by
Midwives without the Supervision of a
Medical Practitioner |
| | CHAPTER VII Conclusions and Recom-
mendations |

Demy 8vo. 10s. 6d. net.

Abscess of the Brain:

ITS PATHOLOGY, DIAGNOSIS AND TREATMENT

By E. MILES ATKINSON, M.B., F.R.C.S.

This monograph is based upon the Jacksonian Prize Essay for 1926 which has been re-written and brought up to date. Comprised in it are both the original researches of the author and a full account of the present-day state of knowledge on this large and important subject. The book, in the arrangement of its contents and the method of their exposition, has been designed to appeal not only to the specialist, who will find accounts of cases, statistics and a liberal bibliography set apart in appendices for special study, but also to the general practitioner and senior student, for whom the text holds information of interest and value presented in a readable form.

*Illustrated with 8 colour plates, 25 photographs,
12 drawings. Large Demy 8vo. 2ls. net.*

MEDICAL PUBLICATIONS, LTD.,
27 Malden Lane, Strand, London, W.C.2

E. & S. LIVINGSTONE Medical Publishers 16 & 17, Teviot Place Edinburgh.

LATEST NEW BOOKS

VITAL CARDIOLOGY. *A New Outlook on the Prevention of Heart Failure*
By BRUCE WILLIAMSON, M.D., Edin., M.R.C.P. Lond. 352 pp. Illustrated
with Diagrams. Price 15s. net. Postage 9d. (1934)

MATERNAL MORTALITY AND MORBIDITY. *A Study of their Problems.*
By Professor J. M. MUNRO KERR, M.D., F.R.F.P.S. Glas. Crown 4to, 400 pp.
Illustrated with maps, plans, charts, diagrams and X-ray plates. Price 25s. net
Postage 9d. (1933)

ACIDOSIS AND ALKALOSIS. By STANLEY GRAHAM, M.D., F.R.F.P.S., and
NOAH MORRIS, M.D., B.Sc., D.P.H., F.R.F.P.S. Crown 8vo, 216 pp. 21 Diagrams.
Price 7s. 6d. net. Postage 5d. (1933)

"BRIGHT'S DISEASE." *A Handbook for Senior Students and Practitioners.*
By J. NORMAN CRUICKSHANK, M.D., F.R.F.P.S. Glas. Demy 8vo, 220 pp.
Price 10s. 6d. net. Postage 6d. (1933)

LATEST NEW EDITIONS.

HANDBOOK OF THERAPEUTICS. By Professor DAVID CAMPBELL, B.Sc., M.D.
Second Edition. Crown 8vo, 464 pp. 72 Illustrations. Price 12s. 6d. net.
Postage 6d. (1934)

COMBINED TEXTBOOK OF OBSTETRICS AND GYNÆCOLOGY. By Professor
MUNRO KERR, Drs. HAIG FERGUSON and JAMES YOUNG, and Professor
JAMES HENDRY, with other Contributors. Second Edition. Royal 8vo,
1,120 pp., with over 500 Illustrations. Price 35s. net. Postage 1s. (1933)

TEXTBOOK OF MEDICINE. Edited by J. J. CONYBEARE, M.D., Oxon., F.R.C.P.
Compiled by fourteen contributors. Second Edition. Demy 8vo, 1,028 pp. Illus-
trated with Diagrams and X-ray Plates. Price 21s. net. Postage 9d.

A HANDBOOK OF MEDICINE (Wheeler and Jack's). Revised by Professor JOHN
HENDERSON, M.D., F.R.F.P.S. Glas. Ninth Edition. Crown 8vo, 650 pp.
34 Illustrations. Price 12s. 6d. net. Postage 6d.

**PRACTICAL METHODS IN THE DIAGNOSIS AND TREATMENT OF VENEREAL
DISEASES.** By DAVID LEES, M.A., M.B., F.R.C.S., D.P.H. Second Edition.
Crown 8vo, 650 pp. 87 Illustrations, with 8 pp. of Coloured Plates.
Price 15s. net. Postage 9d.

Our complete 48-page Catalogue will be sent Post Free on application.

E. & S. LIVINGSTONE, 16 & 18, Teviot Place, Edinburgh

NORTHWOODS, WINTERBOURNE, BRISTOL

This beautiful mansion in fifty acres of secluded grounds was built specially for the
TREATMENT OF MENTAL AILMENTS.

Certified and temporary patients of both
sexes.

A few voluntary patients received in the
Medical Supt's. house.

Separate bedrooms.

Private suites.

Wireless or other concerts.

**Terms from
4 guineas
a week.**

Private golf course.

Thorough clinical, bacteriological
and pathological examinations.

Visiting consultants.

Occupational therapy.

Garden and dairy produce from
farm on the estate.

For further particulars and prospectus apply to JOSEPH CATES, M.D.

**Telephone & Telegrams:
WINTERBOURNE 18**

**'The Victoria' British Sanatorium,
DAVOS, Switzerland.**

*Most up-to-date establishment for treatment
of pulmonary and surgical tuberculosis.*

Terms, including accommodation, full board (including English breakfast), medical attention, nursing, laboratory work, light, heating and baths, from Fcs. 14 per day.

Medical Superintendent:

BERNARD HUDSON, M.D. Cantab., M.R.C.P. Lond.,
Swiss Federal Diploma.

SPECIALISTS=====

ARE ESSENTIAL TODAY

PRINTING of every descrip-
tion must be designed by
Craftsmen who understand
YOUR SPECIAL NEEDS

Printers of Uncommon Skill

HENRY F. THOMPSON & Co.

**UNIVERSAL HOUSE
SOUTHWARK BRIDGE - S.E.1**

Telephone HOP 2523

BETHLEM ROYAL HOSPITAL

Monks Orchard, Eden Park
Beckenham, Kent

For the Reception and Treatment of Cases of NERVOUS & MENTAL DISEASE

President—LORD WAKEFIELD OF HYTHE, C B E., LL.D.
Treasurer—SIR LIONEL FAUDEL-PHILLIPS, B.L.
Physician Superintendent—J. G. PORTER PHILLIPS, M.D., F.R.C.P.
Senior Assistant Physician—MURDO MACKENZIE, M.D.
Junior Assistant Physician—DAVID ROBERTSON, M.D.
Pathologist—CLYMENT LOVELL, M.C., M.D.

CONSULTING STAFF

Surgeon—STANFORD CADE, F.R.C.S.
Gynaecologist—THOMAS GEORGE STEVENS, M.D., F.R.C.S.
Ophthalmologist—J. D. MAGOR CARDELL, M.B., F.R.C.S.
Laryngologist—W. MAYHEW MOLLISON, F.R.C.S.
Anaesthetist—CECIL M. HUGHES, M.B., B.S.
Dental Surgeon—ARTHUR BULLEID, M.R.C.S., L.D.S.
Neurologist—C. C. WORSTER DROUGHT, M.A., M.D., M.R.C.P.
Radiologist—E. ULYSSIS WILLIAMS, O.B.E., M.R.C.S., L.R.C.P.
Director of Psychological Dept—WILLIAM BROWN, M.A., M.D., D.Sc., F.R.C.P.
Assistant Ditto—R. J. BARTLETT, M.Sc.
Director of Physiotherapy Dept—ALASFAIR MACGREGOR, M.B., C.M.

Patients of the educated classes, in a presumably curable condition, are eligible for admission, and those who can contribute 5 guineas weekly towards the cost of treatment and maintenance may be received as vacancies arise. The Committee will also consider applications for admission at lower rates and in certain cases will be prepared to admit patients free of charge. With a view to the early treatment of eligible cases Voluntary or Uncertified Patients are admitted.

For forms or further particulars apply to the Physician Superintendent.

ROTUNDA HOSPITAL DUBLIN.

UPWARDS of 3,000 maternity cases and 1,000 gynaecological intern patients are treated in the Hospital during the year. Besides the Hospital there is an extern Maternity Department with over 2,000 cases. The routine for Students consists of attendance at the Morning Lectures on Midwifery and Gynaecology, examination of patients in the Gynaecological Department, attendance at operations and all abnormal labour in the Hospital Wards, and conduction of labour cases in the intern and extern departments.

In addition there is a large Antenatal Clinic and an Infants' Department where students are encouraged to attend. The Pathological Laboratory is open to the Class, and the X-ray plant adds greatly to the Hospital. A Biochemical Laboratory will be open in the near future.

Qualified Students are given facilities for following and observing all abnormal cases in the hospital or district, and are allowed, so far as possible, to assist at gynaecological operations.

The Hospital Courses are always going on during the year, and Students can join at any time. The class is limited, therefore it is advisable to register in advance. Excellent residential quarters and Three Tennis Courts and a Standard Squash Racquets Court are available in the hospital.

Extra classes in gynaecological diagnosis and operative midwifery are conducted by the Assistants to the Master.

Fees: One month, £6 6s.; months other than the first, £4 4s. Three months, £12 12s. 1. M. Course £21.

The L.M. Certificate is given to fully qualified Practitioners of Medicine on examination after six months' attendance at the Hospital.

FULL PARTICULARS FROM

A. H. DAVIDSON, M.D., F.R.C.P.I., F.C.O.G.,
MASTER, ROTUNDA HOSPITAL

UNIVERSITY COLLEGE, DUBLIN Medical School

PROFESSORS AND LECTURERS.

ANATOMY

Professor—Edward Patrick McLoughlin, B.A.,
M.B., B.Ch., B.A.O.

PHYSIOLOGY.

Professor—James M. O'Connor, B.A., M.D.

ZOOLOGY.

Professor—J. Bayley Butler, M.A., M.B.

BOTANY

Professor—Joseph Doyle, B.A., D.Sc.

CHEMISTRY.

Professor—Thomas J. Nolan, B.A., D.Sc.

Lecturers—Joseph Algar, M.A., D.Sc.

John Keane, M.Sc., Ph.D.

EXPERIMENTAL PHYSICS.

Professor—John J. Nolan, M.A., D.Sc.

Physics Lecturers—John J. Dowling, M.A.

Patrick J. Nolan, D.Sc.,

Ph.D.

PATHOLOGY AND BACTERIOLOGY.

Professor—T. T. O'Farrell, F.R.C.S.I., D.P.H.

Lecturer in Special Pathology—W. M. Crofton,

B.A., M.D.

PUBLIC HEALTH AND BACTERIOLOGY.

Professor—W. D. O'Kelly, B.A., M.D., D.P.H.

MEDICINE.

Professor—Henry F. Moore, M.D., D.Sc.

SYSTEMIC MEDICINE

Professor—James N. Meenan, M.B., D.P.H.

SURGERY.

Professor—Henry L. Barniville, B.A., M.D.,
M.Ch.

SYSTEMIC SURGERY.

Professor—Henry S. Meade, F.R.C.S.I.

MIDWIFERY AND GYNAECOLOGY.

Professor—Robert P. Farnan, M.B.

GYNAECOLOGY.

Lecturer—Reginald White, F.R.C.S.I.

OBSTETRICS.

Lecturer—James J. O'Kelly, B.A., M.B.,
B.Ch., M.A.O.

MATERIA MEDICA AND THERAPEUTICS.

Professor—Theobald W. T. Dillon, M.B.,
B.Ch., B.A.O.

OPHTHALMOLOGY.

Professor—Louis Werner, M.B., F.R.C.S.I.

MEDICAL JURISPRUDENCE.

Lecturer—John McGrath, M.Sc., M.B.

DENTAL SURGERY.

Professor—E. Sheridan, F.R.C.S.I., M.D.S.

DENTAL MECHANICS.

Professor—J. I. Potter, L.D.S., M.D.S.

ORTHODONTIA.

Lecturer—Laurence Flanagan, B.D.S., M.B.,
B.Ch., B.A.O.

The Professors are assisted in the principal departments by a staff of Demonstrators. Regulations for Courses of Study for Degrees, General Regulations containing information as to Scholarships, etc., may be obtained from THE REGISTRAR, University College, Dublin.

THE COTSWOLD SANATORIUM

Medical Superintendent
GLOFFREY A. HOFFMAN,
B.A., M.B., T.C. Dub.

Assistant Physician
MARGARET A. HARRISON,
M.B., B.S. Lond.

Consulting Laryngologist
SIDNEY BERNSTEIN,
M.R.C.S. Eng., L.R.C.P. Lond.
(Attends regularly)



Telephone, 81 & 82 WITCOMB

Telegrams, "HOITMAN, BIRDLIP"

FIRST opened in 1898 and rebuilt in 1925. On the Cotswold Hills, seven miles from Cheltenham, for the treatment of Pulmonary and all other forms of Tuberculosis. Aspect S.E.W. sheltered from North and East, elevation 800 feet. Pure bracing air. *Special Treatment by artificial Pneumothorax* (X-ray controlled), *Tubercular and Ultra Violet Rays* is available, when necessary, without extra charge. X-ray plant. Electric light. Radiators, hot and cold basins, and Wireless in all rooms. Up-to-date main drainage.

Apply THE SECRETARY, THE COTSWOLD SANATORIUM, CRANHAM, GLOUCESTER

Full Day and Night Nursing Staff.

Terms 4½ to 7 Guineas.

CAMBERWELL HOUSE

33, Peckham Road, London, S.E.5

Telegrams
"Psycholia, London"

Telephone
Rodney 4731 2.

For the treatment of **MENTAL DISORDERS.**

Also completely detached Villas for mild cases, with private suites if desired. Voluntary Patients received. Twenty acres of grounds. Hard and Grass Tennis Courts, Putting Greens, Bowls, Croquet, Squash Racquets, and all indoor Amusements, including Wireless and other Concerts. Occupational Therapy. Callisthenics, and Dancing Classes. X-ray & Actino Therapy. Prolonged Immersion Baths, Operating Theatre, Pathological Laboratory, Dental Surgery & Ophthalmic Department. Chapel.

Senior Physician: Dr. HUBERT JAMES NORMAN

Assisted by three Medical Officers, also resident, and visiting Consultants.

An Illustrated Prospectus, giving fees which are strictly moderate, may be obtained upon application to the Secretary.

* * *

The Convalescent Branch is HOVE VILLA, BRIGHTON, and is 200 ft. above sea level.

ADVERTISEMENTS

LIQUOR PEPSINÆ

(LONG)

A most concentrated and effectual Preparation of Pepsine ;

Invented by the late Dr. Edward Long of this firm.

EXTENSIVELY PRESCRIBED FOR THE PAST SIXTY YEARS BY
LEADING MEDICAL PRACTITIONERS.

It is sweet and palatable even for children.

DOSE. 3ss to 3j at or immediately after meals.

Each batch is carefully tested.

Hamilton, Long & Co. feel warranted by the approbation of the leading medical men for a number of years, in recommending this preparation with confidence. As the faculty generally may not be aware of it, Hamilton, Long & Co. beg to add that the original idea of making an efficient preparation of Pepsine was suggested by the late Dr. Edward Long, of this firm.

An account of the Liquor Pepsinæ was given in "The Medical Press" of the day, and Dr. E. Long sent to a Pharmaceutical Congress held in England at that time, specimens of digested chops, steaks, etc., with a second interesting series demonstrating the part played by Pancreatine in the digestion of fats.

Liquor Pepsinæ cum Bismutho Co.

(LONG)

FORMULA: Each drachm contains, in addition to "Long's Liquor Pepsinæ" cum Bismuth, "Morris' Mur.", 1/24 gr., Acid. Hydrocyanic B.P., 2 mins., and Tinct. Nucis Vom., 5 mins.

Dose. One drachm in a little water just before or after meals, twice or three times daily.

Liquor Pepsinæ cum Bismuth

(LONG)

FORMULA: Each fluid drachm contains a concentrated form, besides "Liq. Pepsinæ (Long)," a full dose of Liquor Bismuthi B.P.

Dose: One teaspoonful in water shortly before meals.

FOR 108 YEARS
HAMILTON, LONG & CO., LTD.

have served the Profession as **Prescription Specialists** and have been patronised by successive generations of Medical Practitioners. They carry a large and comprehensive stock of Medical Products, Sera, Vaccines, etc. Oxygen Gas available at the different establishments.

5, LOWER O'CONNELL STREET, DUBLIN

Branches at 107, Grafton Street, Dublin ; 1, Rathmines Terrace, Rathmines ; and 35, Upper George's Street, Dun Laoghaire.

